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PROSTATIC URETHRAL ADENOMA: ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS, HISTOLOGY AND PATHOLOGY.

By M. GRAHAM SUTTON,
Brisbane.

FOR many years there has been great controversy as to the nature and point of origin of so-called prostatic hypertrophy, and there has been still greater confusion as to the cause of it. In this article and another to follow I wish to bring to the notice of the medical profession some considerations, factual and otherwise, concerning obstruction of the bladder in elderly men and its relief by operation. In order that those interested may fully grasp the reason for my caption and the modern operative procedure that it entails, it will be necessary for me to present the latest conception as to the anatomical origin of this obstruction.

It is not my purpose to consider the cause of it fully, and before I discuss its nature and point of origin it will be necessary to outline the anatomy of the prostate gland and touch on the physiology of micturition, as well as to say something about the histology and pathology of the condition.

ANATOMY OF THE PROSTATE AND PHYSIOLOGY OF MICTURITION.

Anatomy.

The prostate gland and prostatic portion of the urethra are composed of glandular tissue, with which are associated numerous ducts supported by a muscular and fibrous stroma. The number and relationship of these elements have been variously described in standard textbooks from time to time. Much anatomical research, both macroscopic and microscopic, has been carried out; but little of this comes to the notice of the medical profession as a whole, and even if it did, there would be found no unanimity of opinion on the histology of the glandular elements and their ducts. Consequently, so as not to burden you with the details of the various researches on the subject, it will best serve our purpose if I state the anatomical conclusions in a broad way.

The prostate is a gland that has many vicissitudes. It starts life as five lobes—two lateral, an anterior, a posterior and a middle lobe—according to the embryological researches of Lowsley (1912). Johnson confirmed these observations embryologically, and most American urologists accept them.

The anterior lobe soon falls by the wayside owing to atrophy of its rudimentary gland acini and tubules. The middle lobe never reaches maturity, and takes the form of a prespermatic or posterior commissure, while the lateral lobes develop to manhood and enter active service as a double-barrelled organ. This has always been the contention of Hirsch (*The Journal of Urology*, June, 1931, page 669). As old age approaches they are retired to the reserve list, and the glandular tissue atrophies and fibrous tissue makes its appearance.

However, three (and sometimes four) more spurious "lobes" may make their appearance with the advent of so-called prostatic hypertrophy in a certain number of men. These are superimposed on the existing relics and are commonly called enlarged middle and lateral lobes, with in some cases an anterior lobe in addition. If surgical treatment is instituted, these so-called enlarged lateral and middle lobes are enucleated and the prostate is left as a shell of prostatic tissue—the "compression capsule"; this consists of the flattened original lateral and doubtful posterior lobes.

Stated more scientifically, the studies of specimens of prostates removed at autopsy in the various decades have revealed varying amounts of glandular tissue, muscular tissue and fibrous tissue. These variations are dependent on two factors, namely, (i) that of time, and (ii) that of location.

(i) In the infant and the aged, the proportion of glandular tissue is relatively small. This is not true just after adolescence and in early adult life, when there is present a larger proportion of glandular tissue than of muscle and fibrous tissue. After the age of forty years, the glands in the lateral lobes become somewhat dilated and cystic. As the years accumulate, the activity of these glands is less vigorous, with the result that in old age the epithelium of the glands is definitely diminished in height and their secretory action is lessened. The prostatic gland should normally atrophy in elderly subjects, as do all other glands.

(ii) In the normal prostate gland, the relative amounts of glandular and fibrous tissue vary in different locations. For instance, at the bladder neck the muscular wall of the urethra is well developed and thick, and is relatively free from glands and ducts. Near the verumontanum the muscular wall of the urethra is pierced by a large number of ducts, and there are some glands in it.

There are other facts relating to the anatomy of the prostate, the bladder neck and the first part of the urethra which must be considered.

Essentially the bladder consists of interwoven bundles of smooth muscle fibres, which may for the purpose of description be divided into three layers—outer longitudinal, middle circular and inner longitudinal. The former two go to form the internal sphincter, with which I shall deal later.

The prostate surrounds the first part of the urethra and by virtue of its fusion with the base of the bladder affords firm fixation for the proximal end of this part of the urethra. The prostate in turn is slung in its fascial sheath, which posteriorly is a fused composite structure, including the retroprostatic portion known as the fascia of Denonvillier, developed in two layers.

The prostatic portion of the urethra is carried at an angle, convex forwards to the general axis of the bladder, and the point of this angle is at the internal sphincter or bladder neck. From this point the urethra—a collapsed tube—with unstriped muscle walls describes a gentle curve, concave forwards until it reaches the deep layer of the urogenital diaphragm (superior perineal fascia, really part of the intrapelvic fascia).

The prostatic part of the urethra is about one and a half inches in length and is embedded in the prostate, somewhat nearer to the anterior than to the posterior surface. It is the widest and also the most dilatable part of the urethra. About half an inch wide at its mid-point, it narrows slightly at each end; but it may be dilated so as to permit the passage of the finger into the bladder. On account of its distensibility, the removal of stones from the bladder was at one time carried out through the prostatic portion of the urethra. A longitudinal ridge, termed the urethral crest, occupies the floor of the prostatic portion of the urethra in nearly its whole extent. Longitudinal grooves lie one on each side of the crest; they are termed the prostatic sinuses and receive the prostatic ducts. Near the middle of the crest, on the verumontanum, a small median opening leads into a blind diverticulum, which passes upwards and backwards below the "middle lobe" of the prostate. This is termed the prostatic utricle (*sinus pocularis*), and near its orifice the prostatic portion of the urethra receives the openings of the ejaculatory ducts. There is a short length of the first part of the urethra between the apex of the prostate and the superior perineal fascia—an important and almost forgotten surgical subdivision, to say the least.

The membranous part of the urethra is narrower than any other part of the urethra except the external meatus. It is only three-quarters of an inch long, and it lies between the two fascial layers of the urogenital diaphragm, surrounded by its sphincter muscle.

The Urethral and the Prostatic Glands.

As early as 1894, Jores, and later in the same year Loeschke and Adrion, described the glands of the prostate as of three catagories: (i) short and simple "mucosal glands" situated on the lateral and anterior walls of the urethra opposite the verumontanum, but also on the posterior wall in the region of the sphincter, where they were more numerous; (ii) longer and more branched "submucosal glands", coursing antero-posteriorly round each side of the urethra, with their ducts emptying in the lateral prostatic sulci; (iii) the principal mass of glands which make up the prostate proper.

In an outstanding article in *The Journal of Urology*, December, 1939, as a result of a beautiful piece of anatomical research, I. E. Le Duc makes the following statements concerning the prostatic portion of the urethra:

The urethral crest, which divides the urethra in the mid-line posteriorly, is generally referred to as the verumontaum. Actually the verumontanum forms only the middle portion of the crest, which tapers from the superior edge of the verumontanum towards the neck of the bladder to form a more or less flattened superior crista. Below the verumontanum, running as far as from 1·5 to 2·0 cm. distally, is found a very definite narrow elevation, the inferior crista, easily demarcated from the verumontanum itself.

The ductal orifices are exceedingly variable in size, grouping and location, and may be obscured by œdema or granulations. Occasionally dilated, many are but pinpoint in size and do not by their size denote the amount of granular tissue drained. They are found most numerously in the prostatic sinuses, but scattered orifices are seen on the lateral walls of the urethra and on top of the superior crista. There is also almost uniformly a line of ductal orifices on the lateral aspect of the verumontanum itself, posterior to the orifices of the ejaculatory ducts, ascending the verumontanum toward the inferior crista, on the summit of which the line ends. Ducts may be found opening on the lateral walls of the inferior crista, usually close to its summit, as far as from 5 to 10 mm. below its attachment to the verumontanum. Scattered orifices in the urethra distal to the verumontanum are also to be found.

Referring to the lobes of the prostate, Le Duc continues:

The adult prostate is thus seen to consist chiefly of two lateral masses, or lobes, and a small median lobe, which may occasionally be replaced by a pre-spermatic commissure. An anterior lobe was not demonstrated, but ducts from the lateral lobes extend quite frequently up to, or even into the anterior commissure.

A complete absence, in these adult prostates, of anything resembling a "posterior lobe" may probably be interpreted in one of two ways. Either the embryological posterior lobe becomes atrophic, as does the anterior lobe, in later development; or the post-natal growth of these glands becomes gradually directed laterally. Confusion with the posterior lamella found in prostatic hypertrophy should no longer exist, as this study shows the posteriorly compressed ducts of the lateral lobes and even of the median lobe to compose this posterior lamella, or "surgical capsule" of the prostate. Correlation of the prostatic ducts with the urethral orifices shows that, in general, the duct will drain glandular tissue immediately lateral to the orifice, or in case of the median lobe, immediately proximal. Groups of ducts opening on the verumontanum and inferior crista, draining tissue of the lateral lobes, have been described.

The separate existence of submucosal glands corresponds with existing knowledge as to their location and structure.

Collectively these glands are referred to as the accessory urethral glands.

The glands situated in the mucosa of the prostatic portion of the urethra between the verumontanum and the vesicular orifice are commonly known as the mucosal or submucosal glands of Home. The importance of these glands cannot be over-estimated, since it is from them that the adenomata arise in prostatic obstruction, as will be emphasized in this article.

Homologues of these glands are also found, but not in all cases, beneath the mucosa of the posterior aspect of the vesicular orifice (the subcervical glands of Alberran), beneath the mucosa of the trigone (subtrigonal glands) and even behind the interureteric bar in the lower half of the bladder. These glands are of independent origin, and probably have no normal functional relationship with the prostate gland.

The subcervical group of Alberran, which consists of thirty or more branched tubules whose ducts open in the mid-line of the floor of the urethra proximal to the verumontanum, are of some importance. The tubules grow entirely within the sphincter muscle of the bladder and occupy for the most part the mucosa, some few penetrating into the submucous coat. They differ from the prostatic tubules in that they are simpler in structure and are lacking in a definite muscular and connective tissue stroma. Their importance lies in the fact that they are subject to frequent pathological changes resulting in enlargement and consequent obstruction to the vesicle outlet. In 24% of post-mortem specimens taken from men over thirty years of age, Lowsley found that these glands had enlarged sufficiently to cause demonstrable signs of obstruction in the bladder. The same writer describes a second group of accessory glands, which he calls the subtrigonal group of tubules, as follows:

> They occur in the mucosa of the trigonum vesicæ usually below the central point and are found as far anterior-ward as its apex. They are for the most part simple tubules which extend to the mucosa and somewhat into it. In the younger specimens there are no branches, but some of those found during the middle age period show one or two small branches. There is nothing distinctive about the structure of the members of the subtrigonal group. The mucous lining is composed of transitional epithelium similar in type to the vesical mucosa. The cells are much piled up, in some cases five or six deep. Their lumina are quite small, as a rule. These tubules are of importance on account of two facts: because their position is such that an overgrowth or enlargement from any cause will bring about a disturbance in the emptying of the bladder; secondly, because an enlargement of the group does occur in small percentage of cases. I have observed six non-malignant tumours of the trigonum vesicæ intravitam and three in post-mortem specimens.
>
> The number of these finger-formed tubules increase markedly after birth but are found in the embryo after the fourth month.
>
> More than twenty of them are observed in every specimen older than four years.

Young also has described cases associated with pedunculated tumours originating from the subtrigonal group of tubules, which caused obstruction by blocking the vesical outlet.

Le Duc draws the conclusion that "the glands taking part in the formation of prostatic hypertrophy appeared to be entirely of the sub-mucosal variety".

The Trigone Muscle.

The anatomical and clinical division between the bladder and the posterior portion of the urethra is situated at the internal sphincter; above this line of division is the trigone of the bladder; below it, the superior *crista urethræ*. The trigone muscle, however, courses under the mucosa through both these areas from the interureteric bar to the verumontanum.

The embryological and anatomical studies of Wesson have shown that the trigone muscle is a definite anatomical entity derived from a fan-like extension of the longitudinal muscle layer of the ureters, which is superimposed upon the inner layer of the detrusor muscle. Some of the fibres pass medially to form the *ligamentum interuretericum*, or Mercier's bar; others interlace with the corresponding fibres of the opposite side and others (Bell's muscles) take an oblique course towards the vesical orifice, where the trigone becomes thicker and denser. Continuing over the dorsal lip of the vesical orifice, it spreads out over the dorsal wall of the prostatic portion of the urethra as the internal longitudinal muscle layer. Wesson has further shown that the so-called internal sphincter consists of two loops of muscle, a ventral loop derived from the external longitudinal layer of the detrusor and the dorsal loop having its origin from the circular fibres of this muscle.

Wesson considers that this demonstration of the anatomical relationship of the trigone and vesical sphincter furnishes a rational explanation of the physiology of micturition and holds his view is amply confirmed by clinical observations in the various types of vesical orifice obstruction. From these studies he concludes that the internal sphincter, except during micturition, is tightly closed by the tonicity of the detrusor, from which its circular and longitudinal loops are derived. The act of micturition, he believes, is accomplished by a simultaneous contraction of the detrusor and the trigone. During contraction the trigonal muscle shortens and broadens in such a way as to depress and widen the posterior 90° of the sphincter, thus converting it into an irregularly triangular opening.

The arc made by the course of the trigonal muscle through the sphincter into the prostatic portion of the urethra is greatly shortened during contraction, the verumontanum often being brought to occupy a momentary position just outside the internal sphincter.

Trigonal activity thus resolves itself into a contraction which forcibly depresses and widens the floor of the sphincter and thus causes it to open.

Macalpine, however, has pointed out that since McCrae has shown that the trigone muscle is a thin sheet whilst the internal sphincter is a definite structure, and since there are many elastic fibres helping to close the internal sphincter, it is possible that the sphincter muscle itself relaxes, but that the trigone muscle acting as described above is responsible for holding open these elastic fibres.

More recently there has been a further notable piece of research into the action of this muscle.

In dissection of fixed specimens McMahon (*The Journal of Urology*, April, 1938) found that the fibro-muscular tissue surrounding the vesicles was continued down along the ejaculatory ducts, and that this tissue contained many blood sinuses as described by Testut. Testut, and later Macarthy, described it as surrounding the ejaculatory ducts; but McMahon found it gathered into a stout strand, which lay along the dorsum of the ducts and between them proximally. He makes the following statement:

> Distally this strand became narrower and ended in the fibromuscular tissue at the distal end of the verumontanum. A sheet of tendinous fibres was found to sweep over the terminal portions of the ducts in a fan-like manner. This sheet appeared to be continuous with the tendon of the trigonal muscle, which ends at the proximal end of the veru, and became fixed distally on the fibrous tissue of the urethral floor distal to the verumontanum. This suggested that contraction of the trigonal muscle might have some constrictor action on the ejaculatory ducts at their termination. It was found that the trigonal muscle could be separated by sharp dissection from its attachment in the trigonal area of the bladder and that blunt dissection thereafter readily freed it from the vesical neck and floor of the posterior urethra as far as the verumontanum. From the vesical neck the tendinous fibres spread out fan-wise and traction without further dissection was found to produce straightening of the urethra, some elongation of the verumontanum and approximation of the posterior part of the lateral walls of the urethra towards the verumontanum so tending to narrow or shut off the prostatic sinuses.

Wesson previously had observed that in living subjects the trigonal muscle could be seen by cystoscopy to exert a direct upward pull on the verumontanum.

Experimenting with fresh specimens, McMahon showed that if fluid was flowing in a steady stream from both ejaculatory orifices under 18 inches' pressure, the flow could be stopped by traction on the tendon of the trigonal muscle. In five cases, fresh specimens were fixed on a board and the trigonal muscle was dissected free. The vasa were freed and needles were inserted. Warm gelatine was allowed to flow through these from an elevated container. This soon appeared at the orifices of the ejaculatory ducts and was discharged in a steady stream. Traction on the trigonal muscle stopped the flow in all cases and the muscle was pinned down in this position. The specimen was chilled, fixed and cleared. Examination of these specimens showed that in the last four or five millimetres the ducts, which normally tend to be flattened from side to side in this portion, were so constricted that the lumen here was free of gelatine, while just behind this point the ducts were well filled.

Finally Rose has brought forward a new conception by assigning to the internal sphincter mechanism an intermediate function, "serving as a gate-way to the external sphincter mechanism". In writing of this he makes the following statements:

The external sphincter mechanism extends from the internal sphincter to the inferior layer of the triangular ligament. It resists expulsive contraction of the bladder wall by direct compression, stretch, bend, and disalignment of the internal and external sphincter opening. The internal sphincter is not fixed, while the external sphincter opening is definitely fixed within the layers of the triangular ligaments. Here a circular sphincter muscle contracts, the resultant direct compression being aided by contraction of the deep transverse perineal muscle also within the fascial layers. Upward movement of the perineum elevates the internal sphincter, stretching and further compressing the urethra, and disaligning the internal and external sphincter openings. To initiate voiding, one relaxes the muscles of the pelvic floor, thus lowering the internal orifice, and aligning it with the external sphincter. If the bladder wall involuntarily contracts too strongly, one pulls up, i.e., contracts the perineal muscles; and further one may use the adductor muscles to elevate and compress the internal orifice. This movement, which I have previously designated as "pump handle" in that the urethra is fixed at the urogenital trigone and moves up and down at its free end, which is at the internal sphincter, is brought about in the male by the blending of the peri-prostatic fascia into the fascia of the pelvic floor. Anatomically in a normal young male there is a short length of urethra located between the apex of the prostate and superior layer of the triangular ligament which is free, and which would allow a bending at this point.

When the perineum is in contraction, the anal opening is closed, and when in relaxation, bowel and bladder tend to empty at the same time.

We are all familiar with the arrangement of the perineal musculature, which makes this co-ordination exist, and all of which has the same nerve supply—N. pudendus.

Physiology.

Bearing in mind the anatomical distribution and location of the various muscle layers—involuntary and voluntary—and of the fascial sheaths that cover the latter, to all of which reference has just been made, let us try to formulate a conception of the physiological processes they subserve.

It had always been taught that at the onset of micturition the internal vesical sphincter underwent an "inhibitory dilatation", until Young and Wesson published their discovery that the internal sphincter whose circular fibres are in tonic contraction was actually pulled open by the contraction of the trigone muscle concurrently with the contraction of the detrusor. This view has been widely accepted in America by urologists generally; McMahon in particular has presented strong clinical evidence in support of it.

In England, Kenneth Walker has investigated the view and Macalpine's view of it has already been given (*Proceedings of the Royal Society of Medicine*, November, 1934). Langworthy, Drew and Vest among others, working with cats, have given experimental support of it (*The Journal of Urology*, January, 1940, page 123). Nevertheless there are good reasons for not accepting the mechanism as so positive as Young and Wesson would have us believe. The two most cogent arguments against their contention are, to my mind, first, the fact that an inhibitory dilatation of involuntary muscle sphincters, wherever situated in the body, is too well established; and secondly, the fact that perfect control of the bladder is still possible after "prostatectomy" operations, in which the insertion of the trigone muscle is most surely damaged, provided that the circular component of the vesical sphincter is not interfered with.

Macalpine's view, already quoted, must therefore be nearer the truth and that of Rose very important indeed. In other words, our previous conception of the internal and external sphincters as separate entities has been much too narrow and requires elaboration in the light of the researches referred to.

Correlating all these ideas, I suggest that we compare the urogenital system in the male to a river consisting of a series of reaches with locks between them, and with various tributaries running into them. Comparison should be made, too, with the alimentary canal, with which the system is homologous. In the case of the male there are three reaches and three locks.

The bladder is the first reach into which the ureters eject "water" through a check-valve. The lock to this reach is opened by the relaxation of the vesical sphincter aided by the perineal muscles, as Rose suggests, and by the trigone muscle, as Wesson and Macalpine hold.

It would appear that, while the trigone muscle undoubtedly plays a part in normal micturition, it does not actually pull the sphincter open, but controls its size and shape in the relaxed state, and that it really has more to do with the closure of the ejaculatory ducts during its contraction—that is, during micturition.

The second reach is the prostatic portion of the urethra "locked" at the external sphincter. Into this reach the ejaculatory ducts slowly and steadily eject semen when the "ejaculatory valve" is left patent by the resting position of the trigone muscle (McMahon, Macalpine). During micturition this reach is widened by the passage of the urine and by relaxation of the smooth muscle in its walls and of the voluntary musculature of the perineum. During orgasm it is possible that semen relatively slowly emitted by the smooth muscle of the vesicles is in turn steadily and less slowly injected through the second lock into the third reach—namely, the bulb of the urethra—and accumulating here, each quantum is forcibly and quickly ejected by the perineal muscles, just as are the last drops of urine at the end of the act of micturition.

For this ejaculation, of course, it is necessary for the second lock to close temporarily while more semen is being emitted into the second reach. However, one must not forget that in unnatural "emission" (nocturnal emission, or emission in the later stages of impotence), ejaculation as such is largely absent, and that in this the voluntary musculature of the perineum and external sphincter is inactive. Emission, then, takes place by the action of the involuntary musculature of the vesicles and of the wall of the prostatic urethra, through a partially erected organ (Semans and Langworthy, *The Journal of Urology*, December, 1938).

The third lock is the narrowing of the *fossa navicularis*, which is of the nature of a hose nozzle; this is made to point downwards by the pull of the frenulum. The external sphincter is of a similar nature, but adjustable, and then only in one direction—by tightening (Denny Brown and Robertson). The ducts of Cowper's gland open into the third reach.

When a "prostatic" adenoma is removed, the second reach is "blasted" and its form and function are upset. The first lock becomes of paramount importance, and if it fails the second lock may offer some control provided it is not damaged also, or, what is more likely, nullified by the stiffening effect of reactionary œdema. The latter is probably always present for a time after "prostatectomy", by whatever means procured. If the method employed is so rough and destructive as to break down the vesico-urethral angle or seriously tear the circular component of the internal sphincter, incontinence of urine results through a "pipe-stem" membranous urethra (second lock, external sphincter). As the reactionary œdema subsides the temporary incontinence of urine clears up, provided there has been no gross damage to the first lock. If there has been gross damage to the first lock, permanent incontinence of urine is the result, though it may be only a stress-incontinence.

Prostatic operations must therefore be designed to avoid gross damage to the first lock. That is why Harry Harris's operation is so successful and why suprapubic enuculation by the intraurethral method in the case of an easy enucleation is seldom followed nowadays by permanent incontinence of urine. It occurs only in the bungled, difficult suprapubic operation. Gross damage to the internal sphincter would appear also to be the reason why incontinence of urine has in the past been more frequent in the perineal operation.

In 1941 the deft intracapsular enucleation of Young and the other first class American perineal operators—Hinman, Lowsley, T. Gibson, and particularly Belt, Johnson and McCarthy—avoided this nasty complication of an otherwise anatomically perfect operative procedure. By their operative skill and careful technique and their respect first of all for the external sphincter, and during

later months for the all-important internal sphincter, these workers have obtained better results and have revived the perineal operation and put the "spotlight" on it.

Histology and Pathology.

It has been the custom for years in text-books and journals, in discussing enlargement of the prostate, to speak of hypertrophy of either one or both lateral lobes with or without concurrent enlargement of the median lobe. For a long time palpation *per rectum* was the chief means of ascertaining the size of the prostate and of discovering the relative size of the "lateral lobes". Enlargement of the median lobe was diagnosed clinically by shrewd diagnostic weighing of the symptoms together with the information (of obstruction) obtained by the sound.

With the advent and growth in popularity of the cystoscope a nice differentiation of the distribution of the elargement was made possible and other causes of obstruction to urination were discovered. The *prostatisme sans prostate* of the French (fibrous prostate with median bar) was proved and more easily diagnosed by this means.

Obstructions due to pedunculated tumours of the trigone or to hypertrophy of the trigone itself were discovered, and so forth. In addition much smaller enlargements of the "lateral" or median lobes became detectable, and at a much earlier stage of their development, and urologists began to have a new awareness about intraurethral or intravesical enlargement of the lobes of the prostate or of the middle lobe as the case might be. And finally, a greater distinction was made between protrusion of the middle lobe through the *sphincter vesicæ* and enlargement of the lobe beneath the trigone.

Discussions took place as to the bearing of all these various types of enlargement upon the symptoms and signs of the disease and upon their effect on the bladder wall (such as hypertrophy or dilatation and thinning) and upon the amount of residual urine. At operation the glandular masses were found to be adjacent to the urethra and to be covered only by a thin layer of tissue—the mucosa—and of thinned-out circular muscle fibres of the internal (vesical) sphincter.

Suprapubic enucleation of these glanduar masses could be achieved, either by tearing through or incising the base of the bladder over the lobe bulging into the bladder, or alternatively by passing a finger into the urethra and splitting what was thought to be the anterior commissure between two lateral lobes. The latter is still the better position in which to commence the enucleation when performing so-called suprapubic prostatectomy, because it avoids injury to the internal sphincter; but it is not the anterior commissure that is split.

Because the histological study of the surgical specimens thus removed demonstrated their glandular nature, it has been assumed for a long time that their origin was the glandular tissue of the prostate itself. Indeed, this is the idea to which the large majority of members of the medical profession still adheres; one must admit they have no reason to think otherwise, because that is what they have been taught and what they read in the literature available to them. Even the majority of urologists still cling to this idea, despite the fact that seekers after the truth have from time to time brought much evidence to refute it.

Histology.

In 1902 Alberran and Motz discovered that hypertrophy of the so-called middle lobe was derived from the submucosal glands, and in 1905 Motz and Pereareau went further and stated that all hypertrophies of the prostate, so-called, arose from these glands. Jacoby, in 1923, confirmed the work of Loeschke and Adrion and showed photomicrographs of small and early nodules or spheroids of hypertrophy just beneath the urethral mucosa with ducts still communicating with the urethra.

Alberran, Zukerkandl, Papin and Verilac, Ribbert and many others have confirmed the original contention of Motz and Pereareau, the majority of authors on the subject have supported it and Hugh Hampton Young, the American father of urology, says "there is now no longer any reason to doubt its essential truth".

Nevertheless many competent pathologists and urologists could not quite bring themselves to accept the urethral origin of "prostatic hypertrophy", and in 1938 we still find Boyd in his text-book of pathology stating that "the lateral lobes may be enlarged or a new middle lobe may be formed by hypertrophy of the group of glands which lie in the floor of the urethra."

Many urologists still agree with Marion in his contention that it is illogical to deny the prostate glands proper the ability to form "adenomata".

Nevertheless, clinical surgery upholds the idea as Young puts it in his text-book; in support of it he shows beautiful cross-sections taken from Henry Wade's work.

The conception of Motz and Pereareau explains perfectly the findings in hypertrophy of the prostate. Since the enlargement always begins in the "inner" or mucosal glands, it is always central, in close relationship with the urethra, and surrounded by a layer of prostatic tissue, normal except for the result of compression. Since these prostatic glands proper all empty into the posterior aspect of the urethra (Fig. 226) they tend to be spread out over the posterior and lateral surfaces of the hypertrophied masses, forming the "posterior lamella" as seen at operation or autopsy. Thus the posterior lamella, with the ejaculatory ducts within it, does not represent a "posterior lobe" of the normal prostate, but rather the entire normal prostate, distended and thinned out by the growth of the hypertrophied masses within it (Fig. 227).

As the hypertrophied masses enlarge, their development varies greatly according to the point at which the initial enlargement occurs. If the process begins in the region of the vesical orifice (Fig. 229) the line of least resistance must be upward through the sphincter, and as the mass increases in size it pushes into the cavity of the bladder (Fig. 230). In its growth it is influenced by the sphincteric and trigonal muscles which, as we have shown, hypertrophy as a result of the increased work put upon them in opening the vesical orifice by the increasing growth of the prostatic obstruction. The frequent contractions of the sphincter cause a groove to appear in the mass as it enlarges so that, eventually, as the intravesical portion increases in diameter the whole will have a pedunculated appearance. If the growth gets very large, the sphincter may be so widely dilated that it atrophies and eventually practically disappears. If the growth pushes up exactly in the mid-line, it is probable that the trigonal muscles (muscles of Bell) passing over or around it, play an important part in determining its shape and account for the bi-lobed appearances sometimes seen.

This is referred to by Macalpine and very well portrayed in his article in *Proceedings of the Royal Society of Medicine* (quoted above).

Young continues:

The intravesical mass is usually posterior, but occasionally may arise anteriorly (Fig. 231) apparently from the ventral group of glands, which are short and belong to the same type as the suburethral ones. In still other cases the growth may extend almost or quite around the entire urethra, so that a structure shaped like the uterine cervix pushes up into the bladder. When mucosal glands are present on the trigone, or even beyond the inter-ureteric bar, they may also hypertrophy, causing the intra-vesical masses. Sometimes this occurs in younger individuals, the resulting "cystic adenoma" markedly resembling prostatic hypertrophy under the microscope (see Neoplasms of the Bladder).

The intra-vesical type of growth is the most apt to produce obstructions as it pushes up through the sphincter. If the mass develops from gland groups at or above the sphincter, as it becomes pedunculated it may fall into the orifice during micturition and produce obstruction in the manner of a ball-valve while still very small (Fig. 230).

If, however, the growth begins further down the urethra, it increases laterally and shows no tendency to push through the sphincter, presumably because it becomes too wide before its upper margin reaches the sphincteric region. This form of growth is much more apt to extend around the urethra, but is usually seen as two masses, lateral to the urethra (Fig. 228). This suggests origin from two different foci. The two masses may be equal in size, but one may be larger than the other, or, rarely, the growth may be confined to one side. Posteriorly, the masses are separated by the posterior lamella. They press upon the urethra, converting it into a narrow slit, with its long diameter extending

antero-posteriorly. This slit may extend as far anteriorly as do the lateral masses, or they may come in contact with each other in front of it. If the ventral group of glands take part in the growth there may be a layer of hyperplastic tissue running across in front, but more usually this does not occur, and the lateral masses are united, anteriorly to the urethra, only by a thin sheath of fibrous tissue. It is through this sheath that the finger breaks in the first stage of a suprapubic prostatectomy.

Yet we still talk of prostatic hypertrophy!

Pathology.

So much for the anatomical place of origin of so-called prostatic enlargement. Now let us consider the changed views as to its histological point of origin.

One hundred years ago Velpeau, and in 1873 Thompson, recognized microscopically that in sections of the prostate gland there were fibro-muscular masses and glandular masses. More recent workers have made similar studies, and have recognized glandular and aglandular masses and have found them to be coexistent in the same specimen.

In 1925 a great step forward was made when Reischaur presented his new theory and gave us a real insight into the histological tissue which actually commenced the formation of the nodular masses or spheroids. This was that the essential lesion of prostatic hypertrophy was the nodular proliferation of fibrous tissue into which glandular penetration and growth took place only as a secondary phenomenon from the periphery of the nodule. Then Deming and his co-workers came along, but not until 1939, and abundantly confirmed Reischauer's contentions. He studied 29 prostates, virtually normal in size, taken from over 200 collected for the purpose.

Glandular and aglandular nodules were found present in most specimens existing together. In six there were only aglandular masses; but in none were glandular masses found without aglandular masses. This fact pointed to the conclusion that the one developed into the other as Reischauer had demonstrated.

The aglandular nodules were found within the muscular layer of the prostatic urethral wall. They were either single or multiple and located anywhere along its length. The glandular nodules were likewise found in the muscular wall of the urethra, and none were found in the lateral and "posterior" lobes proper. They were variable in size and were surrounded by circular fibro-muscular tissue. The primary aglandular nodule (fibro-muscular), which Deming considers is a hyperplasia about a blood vessel, stimulates a hyperplasia of nearby glandular epithelium; this invades the nodule and produces a glandular mass similar in appearance to the glands in the normal prostate; hence the confusion in the past.

When these nodules increase in size they distort the urethra and compress the true prostatic tissue. This has been shown by many workers. Strangely enough, Deming considers that they do not develop from the subcervical glands of Alberran. He also stresses the fact that they proliferate so as to develop at right angles to the mucosal glands and therefore parallel to the longitudinal muscular fibres of the prostatic urethral wall, and not along the ducts, as would be the case were the lesion inflammatory in origin.

I submit that therefore they must be regarded as adenomata, not of the prostate, but of the prostatic portion of the urethra. This is not a distinction without a difference.

Then came Le Duc's superb piece of research, the anatomical conclusions of which I have quoted. His histopathological findings are in accord with those of Reischauer and Deming. He states that the glands taking part in the formation of the earliest lesions of benign hypertrophy appeared to him to be entirely of the submucosal variety, and that the essential lesion was a nodular proliferation of fibrous tissue.

Although at the commencement of his article Le Duc criticizes the sceptical ones, even Hinman, for their lingering doubts as to the point of origin of so-called prostatic hypertrophy, he ends in the true spirit of a research worker by doubting what he himself set out to prove and by suggesting further studies before the theory he supports is conclusively accepted.

At all events, all the work done goes to show that in the majority of cases, if not all, in which enlargement of the prostate gland is palpable *per rectum* and in which the lesion is considered a benign condition, the tumour (adenoma) producing it is essentially urethral in origin and position.

Those benign tumours of the prostate which are not adenomata of the prostatic urethra may be regarded for the present as one of the following types: (i) fibromyomata (composed of muscle cells and fibrous tissue arranged similarly to the intraglandular stroma of the prostate proper and having scattered glandular elements within and between these masses); (ii) true leiomyomata (composed of interlacing bundles of unstriped muscle fibres devoid of glandular elements). Both of these are rare.

The Cause of Prostatic Urethral Adenoma.

The cause of prostatic urethral adenoma is still undecided. There are now only two theories worth considering, (i) the inflammatory and (ii) the endocrine.

The fact that infection may occur in the prostate in man, be it pyogenic or gonorrhœal, and that animals, particularly the dog, also suffer from prostatic enlargement and are prone to urethritis, lends support to the inflammatory theory. Then again, there is the similarity in microscopic appearance between sections of prostatic tissue removed at operation and those of so-called chronic mastitis. And finally, the fact that prostatic enlargement in the vast majority of cases, as stressed in this paper, takes origin in the mucosal and submucosal prostatic urethral glands, would appear further to strengthen the evidence in favour of its inflammatory nature. However, in the opinion of Deming, the disposition of the fibrous tissue component of the newly formed tissue at right angles to the gland ducts and not parallel to them rules out infection as a cause.

A great deal of investigation has been done which seems to make it almost certain that the disease is endocrine in origin. Histologically the picture closely simulates so-called chronic mastitis and thyreoid hyperplasia, both of which are admittedly due to hormone stimulation. The injection of œstrin causes enlargement of the prostate in animals (for example, mice, possums *et cetera*), and the enlargement is regarded as the same as that which occurs in man. Enlargement of the prostate occurs in male infants soon after birth, and this is explained as being due to an excess of œstrin of maternal origin in the child's circulation.

The pituitary gland is known to have a governing influence on the development of the germinal epithelium of the testis. This was first put forward by McEwan in 1896. The germinal epithelium in turn is believed to exert an inhibitory influence on the growth of the prostate; that is to say, before puberty its action is not present, and after the age of fifty years its action ceases. Hence it is argued that the prostate enlarges up to puberty and after the male climacteric, but not during the sexual life of the individual. It is believed that the interstitial cells of Ledig act in the opposite way to the germinal cells.

X-ray destruction of the germinal cells allows free action of the cells of Ledig and the prostate enlarges. Castration removes both hormonal sources, and the prostate atrophies.

From these and other observations a theory has been evolved. This theory, which is sponsored by Lower, Engel and McCullagh, may now be stated as follows. The development and maintenance of the prostate is directly due to a substance called androtin, which is secreted by the interstitial cells of the testis. If the testes are removed, the prostate atrophies; this atrophy may be prevented by injections of androtin. The secretion of androtin, in turn, is brought about by the presence in the blood of a substance secreted by the anterior lobe of the pituitary gland. The secretion of this substance is controlled by another blood-borne testicular hormone, in this case one which is elaborated in the cells of the semeniferous tubules. This hormone is called inhibin. Since with age the

activity of the tubular cells decreases, less inhibin is secreted. The hypophysis, thus freed of its control, secretes more actively and stimulates the interstitial cells of the testis to produce more androtin.

Against this interesting theory are two clinical facts: first, that not all men over the age of fifty years have prostatic enlargement, and secondly, that although castration causes atrophy of the normal prostate, it does not reduce the senile enlarged prostate. Deming has carried the latter observation a stage further and has shown that castration does not affect the submucosal glands of the prostatic urethra.

Nevertheless, by way of constructive criticism I would suggest that since the prostate is developed by the outgrowth of five buds from the prostatic portion of the urethra, and since the mucosal and submucosal glands in the prostatic portion of the urethra are homologues of these buds, after the male climacteric these glands undergo a kind of compensatory hypertrophy in a certain proportion of men when the prostate glands proper atrophy; this hypertrophied tissue, being placed as it is, eventually becomes a noxious obstruction to the bladder, in much the same way as the uterine fibroid does at times in the uterus.

In searching for support to this hypothesis I came across the following relevant comment by Muschat, Labess and Meranze ("Gonadal Activity in Prostatic Hypertrophy", *The Journal of Urology*, December, 1938, page 807):

> By means of the creatinine utilization method we followed the gonadal activity from boyhood on until it reaches its maximum at puberty. It is then maintained until the age of 50 when with the advancing years of senility the activity gradually declines. Our study and that of others (Koch, McCahey, Hansen and Soloway) indicates that the gonadal activity in prostatic hypertrophy is not diminished but persists normally as in the normal adolescent. It is possible, therefore, to assume that if the gonadal activity persists in spite of the expected physiological decline, an enlargement of the prostate will ensue. This could be explained as an unnatural response to excess gonadal stimulation of the dormant embryological prostatic anlage rests, the periurethral glands, which have been found to be the site of origin of prostatic proliferation and nodule formation in early hypertrophy.

Bolliger's studies are very interesting and might repay reinvestigation on the point of histological origin of the enlargement. In these studies, after the injection of œstradiol benzoate *et cetera* into possums, he found among other things enlargement of the prostate gland.

Whether prostatic enlargement in man is generally analogous to that found in laboratory animals and whether the point of origin is from muscle tissue (Ewing, *re* uterine myomata, and Deming, *re* prostatic adenomata), from fibro-areolar tissue (Le Duc), or from glandular epithelium, are questions that remain for further research to decide.

LEPTOSPIRA POMONA.

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In 1936 a leptospira was isolated from the blood of a dairy farmer who lived near Pomona in south Queensland (26° 30′ south, 153° east), and who was suffering from a fever of eight days' duration.[1] The leptospira has since been referred to as the Pomona strain.

Experience since then has shown it to be an important cause of human illness. Eighty cases of leptospirosis due to it have been diagnosed as the result of tests at the laboratory of the Queensland Department of Health and Home Affairs. Many others have been diagnosed clinically. The geographical distribution of the cases extends from Innisfail, north Queensland (17° 30′ south, 146° east), to Burringbar, New South Wales (28° 30′ south, 153° 30′ east).

The clinical picture of infection is fairly definite, enabling the diagnosis to be made clinically in many cases. The illness is usually mild; in contradistinction to classical and north Queensland leptospiroses, no cases associated with jaundice or ending fatally have yet been observed. The symptoms are similar to those of the mild leptospiroses of other countries—mud fever, seven-day fever *et cetera*.

Serological investigations by Johnson and Brown[2] showed that the Pomona strain was distinct from the three other Australian strains of leptospiræ which had been isolated at that time, as well as from eight overseas strains. The relationship of the Pomona strain to other leptospiræ was investigated further by Walch-Sorgdrager, Bohlander and Schüffner.[3] Summarizing this work, Walch-Sorgdrager[4] concluded that Pomona was a new and easily distinguishable strain.

The established specificity of the organism, and its importance as a cause of human disease, entitle it to a specific name, and the name *Leptospira pomona* is proposed.

Leptospira pomona is essentially similar in morphology and in staining and cultural characteristics to *Leptospira icterohæmorrhagiæ*, but is distinguishable from it and from other leptospiræ serologically. It differs from many leptospiræ in its comparatively low virulence for men and laboratory animals.

Further descriptions of the characteristics of *Leptospira pomona* will be found in the articles quoted in the references.

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THE DISCOVERY OF A FIFTH AUSTRALIAN TYPE OF LEPTOSPIROSIS.

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Leptospirosis was first proved to exist in Australia in 1934, when an outbreak occurred at Ingham.[1] The outbreak was labelled Weil's disease; but in 1937 Lumley[2] showed that the strains of leptospiræ isolated during and after that outbreak fell into two distinct serological types, neither of which was identical with *Leptospira icterohæmorrhagiæ*, the cause of classic Weil's disease. Lumley called these two types *Leptospira australis* A and *Leptospira australis* B.

Leptospira icterohæmorrhagiæ was isolated in 1937, when Johnson, Derrick and Brown[3] published records of three patients, of whom two were sewer workers. Since then, seven more cases of classic Weil's disease have been diagnosed in this laboratory. Two patients were cane cutters, two were sewer workers, two were cooks, and one was an unemployed painter living near a grain store. All except the cane cutters lived in Brisbane.

In 1936 a strain of leptospira was isolated from a dairy farmer at Pomona, Queensland (26° 30′ south, 153° east)[4]. This was later shown to be a new type,[5] and has been named *Leptospira pomona*.[6] Since its isolation, about 80 cases have been diagnosed. The illness in general is comparatively mild and there have been no fatalities.

These were the only serological types of leptospiræ known to exist in Australia before 1940. Many leptospiral

cultures were obtained, but on investigation they were all proved to belong to existing types. From time to time specimens of serum were received at this laboratory which gave partial agglutination with the known types; but the titre was not sufficiently high to be diagnostic. These results raised the suspicion that another leptospiral type possibly existed; but such suspicions had to await confirmation, which was possible only by the isolation of a new strain.

Isolation of a Fifth Australian Strain.

In July, 1940, leptospiræ were cultured from the blood of a patient (H.M.) by Mr. W. H. Reid at the laboratory of the district hospital, Innisfail, Queensland (17° 30′ south, 146° east). The clinical history of this patient is given later in this report. Serum taken on the seventeenth day of illness failed to agglutinate the four known types of leptospiræ, but agglutinated the new strain of leptospira in a diagnostic dilution. Details of agglutination tests with the new strain are given below (Tables I and II).

TABLE I.
Agglutination Tests with Serum of H.M.

Culture.	Agglutination.
Leptospira icterohæmorrhagiæ	Not agglutinated.
Leptospira australis A ..	Not agglutinated.
Leptospira australis B ..	Agglutinated in a serum dilution of 1 in 10.
Leptospira pomona	Not agglutinated.
Culture from H.M.	Agglutinated in a serum dilution of 1 : 10,000.

TABLE II.
Showing Relationship of Strain H.M. to Australian Type Antiserum.

Antiserum.	Titre of Antiserum.	
	Against Homologous Strain.	Against Strain H.M.
Leptospira icterohæmorrhagiæ	10,000	30
Leptospira australis A	3,000	30
Leptospira australis B	3,000	10
Leptospira pomona	3,000	30

It can be seen that the strain H.M. is a new Australian type having no significant relationship with other types. In addition, the strain H.M. was tested against the following world types of antiserum: Canicola (dog strain), Palembang (East Indies), Bangkinang (East Indies), Akiyami A (Japanese strain of *Leptospira autumnalis*), Baermann (East Indies), Batavia (East Indies), Andaman (Indian) and Moscow (*Leptospira grippho-typhosa*). None gave significant agglutination with the strain H.M. All of these types of antiserum (with the exception of antiserum Canicola) were four or five years old, and although they had been kept in the refrigerator for the whole period, were of doubtful potency. Nevertheless, the original titres of most of them were such that one would not expect a complete loss of agglutinins during the period of storage, and this is indicated by the fact that antiserum Batavia (original titre 1:20,000) agglutinated the strain H.M. in a serum dilution of 1 in 30. The disturbed condition of world affairs rendered it impossible to obtain strains or antiserum from overseas sources, hence it cannot be established that the strain is a new world type. It is definitely a new Australian type.

Pending future classification, it is proposed to name the new type *Leptospira mitis*, because the disease it causes in man appears to be relatively mild.

Distribution of Leptospira Mitis.

Since the isolation of the new type, a total of nine cases has been diagnosed in this laboratory. The disease occurs in both north and south Queensland. Four patients were from the Innisfail district. Two were cane cutters, one was a bullock driver engaged in carting timber, and the other was a dairyman. Of the patients in south Queensland, one was a meat inspector, one was a pig slaughterman, and three worked on dairy farms. Of the two cane cutters, one worked on a sugar cane farm adjoining a dairy farm.

There thus appears to be a definite correlation between the disease and association with cattle and pigs. This correlation has been confirmed by the detection of agglutinins against *Leptospira mitis* in the serum of cattle in south Queensland. Incidentally, agglutinins against *Leptospira pomona* have also been detected in the serum of cattle; in addition, agglutinins have been detected in the serum of pigs slaughtered at the Brisbane abattoir, and *Leptospira pomona* has been isolated from a pig. No agglutination tests have yet been performed on pig serum with *Leptospira mitis*, but it is likely that this type, too, will be found to be endemic in both cattle and pigs.

These investigations on the epidemiology of mild leptospirosis are still incomplete and will not be given in greater detail here. They will form the subject of a later report; but in the meantime those interested in further information are referred to the "Annual Reports on the Health and Medical Services of the State of Queensland" for the years 1937-1938, 1938-1939, 1939-1940 and 1940-1941.

Reports of Cases.

The clinical histories from two typical cases are given below. For details of Case I, I am indebted to Dr. R. Hertzberg, then medical officer, Innisfail District Hospital. I quote Case II by courtesy of the General Medical Superintendent, Brisbane General Hospital.

CASE I.—H.M., a male, aged twenty-three years, was admitted to hospital on June 27, 1940, complaining of abdominal pain and painful lumps in both groins, which had been present for three days. He had been "off colour" for three days. The patient had scalding on micturition, but this symptom had been present for years. He was a bullock driver engaged in carting timber from the scrub, near the Beatrice River, north Queensland.

On examination the patient was flushed and appeared to be in pain. No abnormalities were found on examination of the abdomen or respiratory system. There was a soft systolic murmur at the mitral area. The inguinal glands were enlarged and very tender, and there were numerous abrasions on both legs, some of which were infected.

During the next few days the patient's condition became much worse; he complained of severe headache and of pain and aching in the bones and joints. He sweated profusely. On the eighth day of his illness he felt much better, and convalescence was uneventful until his discharge from hospital on July 8, 1940 (see Figure I).

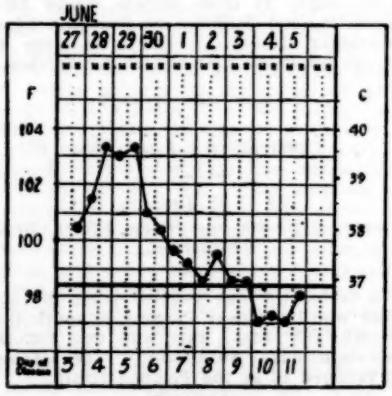

FIGURE I.
Temperature chart of H.M. (Case I).

On the following day the patient was readmitted to hospital, complaining of abdominal pain and vomiting. On examination pronounced tenderness was elicited along the right costal margin; but the liver was not palpable. No jaundice was present. He was again discharged from hospital on July 17.

On July 28 the patient was seen again and complained of occasional headache, vomiting, abdominal pain and pain in the hip region. A fortnight later he stated that he did not yet feel really well.

A number of laboratory investigations were made during the course of his illness. On June 29, 1940, a leucocyte count revealed 6,400 leucocytes per cubic millimetre of blood, of which 85% were neutrophile cells. On the same date five mils of blood were collected in a heparin-tube. After standing for three hours the supernatant plasma was found to contain leptospiræ. On June 30 blood was withdrawn and inoculated into seven tubes of Fletcher's medium. Leptospiræ were seen in all tubes on the seventh day. On July 8 blood was taken for serum agglutination tests. The serum failed to agglutinate known Australian leptospiral types, but agglutinated the strain of leptospira isolated from the patient in a serum dilution of 1 in 10,000.

CASE II.—T.O., a male, aged twenty-eight years, was a meat inspector at the Brisbane abattoir. For the fortnight prior to his illness he had been engaged in inspecting mutton and pork. At 2 a.m. on August 12, 1940, he woke with severe headache and a shivering attack. The headache was mainly frontal and was severe and constant for seven days, when it disappeared. The patient vomited all his food for four days. He had alternate attacks of shivering and sweating. On August 14 he began to suffer from pain in the lower part of the back, which persisted for several days.

The patient was seen by Dr. L. A. Little, who sent him to the Brisbane General Hospital, where he was admitted on August 16. On that date he complained of soreness of the eyes, and stated that he had dull aching in the legs and joints. Physical examination on August 16 revealed that he had a furred tongue, tenderness over the liver, and some congestion of the conjunctivæ.

Fever persisted till August 19 (see Figure II). The patient was discharged from hospital three days later, feeling weak, but otherwise fairly well.

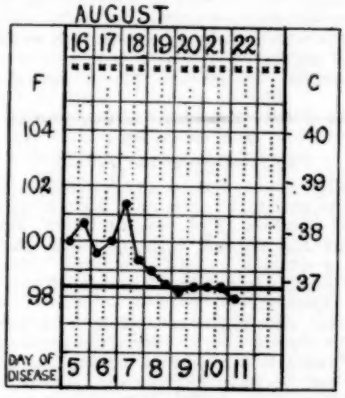

FIGURE II.
Temperature chart of T.O. (Case II).

On August 28 he complained of asthenia and weakness in the legs, and also of dull pain in the loin and at the back of the eyes.

On September 6, after the patient had been at work for three days, he still complained of pain in the sacral region and of generalized weakness. He was "light-headed" if he stood about for too long, and his eyes felt as if he had read too much.

A number of laboratory investigations were undertaken during the course of his illness. On August 17, 1940, blood was inoculated into a guinea-pig. This animal failed to develop signs of disease. The second passage animals also failed to react. On August 19 a blood count revealed 4,790,000 erythrocytes and 8,200 leucocytes per cubic millimetre of blood. A differential leucocyte count showed that 53% were neutrophile cells, 42% lymphocytes, 4% eosinophile cells and 1% monocytes. The hæmoglobin value was 90% and the colour index 0·94. On August 28, four mils of freshly passed urine were inoculated into a guinea-pig by the intraperitoneal route. This animal reacted with fever on the eighth day, and when it was killed showed the post-mortem signs characteristic of leptospirosis. Leptospiræ were seen in a liver emulsion, and were cultured in Schüffner's medium from the heart blood. On examination the strain isolated was found to be identical with *Leptospira mitis*. Second passage animals developed leptospirosis on the fourth and fifth days respectively after inoculation with liver emulsion from this guinea-pig. On August 28 also blood was withdrawn for serum agglutination tests and the serum agglutinated *Leptospira mitis* in a dilution of 1 in 10,000. On September 6 four mils of freshly passed urine were inoculated into a guinea-pig. The animal reacted with fever on the ninth day, and when it was killed showed the post-mortem signs characteristic of leptospirosis.

Comment on Cases.

Helpful diagnostic features are the patient's occupation (for example, association with pigs or cattle), the sudden onset with headache and fever which often waken the patient at night, the persistent headache, the gastro-intestinal upset, and the generalized muscular and joint pains. The conjunctivæ may be congested. Fever lasts about a week. Convalescence is sometimes protracted.

A study of the cases so far indicates that infection with *Leptospira mitis* is indistinguishable clinically from infection with *Leptospira pomona*.[5]

Laboratory tests are essential to confirm the diagnosis and to establish the type of leptospira causing infection.

Acknowledgements.

I am indebted to Sir Raphael Cilento, Director-General of Health and Medical Services in Queensland, for permission to publish this paper and for his encouragement of these investigations. I wish to pay special tribute to Mr. W. H. Reid, laboratory technician, Innisfail District Hospital, for his skill in isolating *Leptospira mitis* and for his generosity in making the strain available for typing.

I am grateful to Dr. E. H. Derrick, Director, Laboratory of Microbiology and Pathology, for much assistance and advice, and to Mr. H. E. Brown, bacteriologist of this laboratory, for his cooperation and his careful attention to leptospiral cultures. Dr. R. Hertzberg kindly supplied the clinical histories of three north Queensland patients.

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SOME ASPECTS OF EPILEPSY.

By N. V. YOUNGMAN,
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IN any clinical disorder there is usually some outstanding feature which dominates our conception of the disease in question. There is the abdominal pain in appendicitis. There is the pneumonia in pneumococcal infection. There is the discomfort or distress in cardiac upset. And there is the major convulsive seizure in epilepsy.

This approach to disease tends to make us forget that the whole organism, rather than one bodily system, reacts to any such disease process. At the same time it has value. It has value because it emphasizes the essential mechanisms of a disease process which are often most clearly portrayed in its major manifestations; it means that if we carefully study these leading features we can better appreciate the changes that are occurring throughout the body. The nature of the abdominal pain reveals not only

the type of change in the appendix, but also its import to the patient, be it anxiety or toxæmia. So I wish to show how a study of the major convulsive seizure of epilepsy can help us to understand the fundamental features of that entity.

Let us first examine the usual description of the major convulsion. It is classically considered in three stages—the aura, the convulsion and the post-convulsion period. The aura is mainly concerned with subjective phenomena; the striking motor features not unnaturally dominate the description of the second stage, while the changes of the post-convulsion period are less well defined. One of the main virtues of this division is its accentuation of the time factor in the development of symptom complexes.

Yet if we pause to consider the severity of the convulsion, it should not be unreasonable to assume that the whole organism, but particularly the nervous system, is involved. This implies not only the accepted organic aspects, sensory or motor, in either an objective or a subjective form, but also those higher nervous features such as memory, mood, orientation, reasoning, intellect *et cetera* which go to complete one's neurological make-up.

In the complete description, it would be desirable to consider how all these different nervous units are affected at each period of the seizure. Then, to make a complete clinical record of this process would be an exacting task. I wish to consider only one small aspect—the changes wrought by this process on memory and behaviour. For reasons which will be apparent later this is not easy in the naturally occurring seizure; but with the advent of shock therapy one has the opportunity of reproducing a comparable phenomenon under conditions which render clinical observation feasible.

I propose to outline certain observations made on the "Cardiazol" convulsion and to consider the significance of these findings in relation to epilepsy and allied clinical conditions.

The Method of Investigation.

The routine method of giving "Cardiazol", together with a general description of the seizure, will first be given. Any additional observations necessary will also be outlined.

A tourniquet is applied to the arm, local anæsthesia is induced, a dose of medicine is given, after which the injection of "Cardiazol" is made. By this method the patient is made aware of certain time intervals. When more detail is required the patient is told beforehand to remember a number, or is asked to count during this period.

From the completion of the injection the various phenomena that follow are timed with a stop watch. This is necessary, because wrong observations on such a dramatic event are frequent unless it is recorded in this fashion.

The earliest sign that the convulsion may ensue is coughing. This occurs towards the end of the injection or soon after it is completed. It is more an indication that the drug has entered the circulation, for it may follow inadequate dosage. In practice it means that a convulsive seizure is imminent.

During this time the patient lies quiet, perhaps a little apprehensive; respirations then quicken in rate and depth before the initial irregular clonic movements of the limbs and face follow. These usually occur ten to fifteen seconds after the injection, and for the next five seconds become more frequent and widespread till the customarily described tonic and clonic phases develop. The tonic stage lasts about seven to ten seconds, and the clonic stage thirty to fifty seconds.

At the end of this period the patient is deeply unconscious. This state gradually lightens, till at the end of five or ten minutes he can be roused and made to answer questions. Should the patient be left undisturbed, he will sleep for a period usually of a quarter of an hour to two hours before resuming his usual activities.

In the cases chosen for detailed investigation, the following series of questions are asked:

What is your name?
Where are you?
Where do you live?
How old are you?
What day is it?
What month is it?
What year is it?
How do you feel?
Where were you born?
Who is the Prime Minister?
Who is the King?
Who is the Premier?
What is 2 times 3?
What is 4 *plus* 5?
Who am I?
Have you ever been sick?
Have you any headache?

They are first asked five or ten minutes after the main seizure ends. The time of asking and the replies given are recorded. This series of questions is repeated at intervals during the two or three hours following the injection. These questions may appear rudimentary and hardly consistent with a survey of a psychological state, but in practice they have served the purpose required. During this period also a realistic toy revolver is shown and the patient is asked to use it.

When the patient appears to have fully recovered from the effects of the seizure, hypno-narcosis is established by an intravenously administered drug, especially in the case of those who have been subjected to the revolver experiment. "Sodium Amytal" and "Sodium Evipan" are used for this purpose. The former produces, from my point of view, a more satisfactory state, and it is also preferred by the patients who have experienced both drugs. During this state the patients are questioned as to the events of the pre-convulsion and post-convulsion periods.

The Results of the "Cardiazol" Convulsion on Memory and Behaviour.

The Pre-Convulsion Period.

In practically every case a pre-convulsion amnesia is present. If the patient is questioned soon after the seizure the period of amnesia is of several hours' duration, but as he regains his normal state of mind the period is stabilized at a quarter or half a minute. His last recollection is of receiving the medicine or of the commencement of the injection.

The persistent amnesia is much longer in certain cases; it may last for several hours. I have not carefully analysed the types of cases in which this occurs, but among them two classes can be defined clinically. First, there is a group of subjects with an intelligence just below normal. The second group is composed of women with clear, fine skins, keen on their homes, conscientious, and of the anxious obsessive type. Only careful memory testing before and after the seizure will reveal the factors governing the length of this period of amnesia.

The behaviour of the person during this period is normal. If the patient has had "Cardiazol" previously and discussed it with others, he may be apprehensive. If the amnesic period is long, the patient has carried on his usual activities up till the time of receiving the injection. Such activities may include his coming to hospital. In those cases in which hypno-narcosis is employed, the events of this period cannot be recollected even when this period is of considerable duration.

The Post-Convulsion Period.

In most cases there is a complete amnesia of one to three hours' duration. This is followed by an incomplete amnesia, and certain memory defects may persist for weeks, or may even become permanent. These defects again are more pronounced in certain persons and if injections are given at frequent intervals.

During the period of complete amnesia, after the initial drowsy and irritable stage has passed, the patient appears perfectly normal and talks naturally on everyday subjects.

Even nurses in close contact with this type of work are unaware of any abnormality in the patient and allow him to go home. The patients have no subsequent memory of how they managed to do this. However, even with the crude test I have used certain defects are present. They may not know their age, place of birth *et cetera*. I do not intend to analyse which components of the memory mechanism are at fault; unless the patients are subjected to these tests, this abnormal state is not recognized.

During this period the subject is amenable to suggestion. He can be made to use the revolver without much persuasion, although I doubt whether my volunteering as the target has any significance. It is unlikely that he would use it spontaneously. When questioned later, the patient will have no recollection of having used the revolver, and will deny having seen it before. No satisfactory account of this period can be obtained when narcotherapy is used. I may mention that in the narco-hypnotic state the patient is less amenable to suggestion, and it is extremely difficult to persuade him to use the revolver.

The Significance of These Observations.

When pathological processes are developing in any part of the body, these changes are reflected in the clinical picture first, and most obviously, in the finer functions of the system involved. Usually these changes are subjective in nature. For instance, slight impairment of appetite, a little more careful discrimination in foods, or a general feeling of ill health often heralds the presence of a more serious bowel disturbance. It is only later that definite syndromes or physical signs develop.

So, too, in the nervous system we would expect to find any acute disturbances affecting most pronouncedly the higher cerebral functions. This has been borne out by the examination of one such function, memory. We have seen that the duration and severity of the different aspects of this pattern vary from subject to subject.

It would not be unreasonable to presume that all the higher nervous functions undergo a comparable disturbance. The duration of this disturbance of these finer functions is prolonged compared with the seemingly essential features, the motor changes. Let us now consider in more detail the significance of these findings.

The Diagnosis of Epilepsy.

Let me first relate the incident of three famous French neurologists, Trousseau, Esquirol and Calmeil. They were walking in the Asylum of Charendon, discussing the diagnosis of feigned epilepsy. Esquirol maintained that even persons thoroughly familiar with the disease could not perfectly imitate it. Suddenly Dr. Calmeil fell down on the floor in violent convulsions. After examining him for a moment Esquirol turned to Trousseau, exclaiming: "Poor fellow, he is epileptic!" But he had no sooner said so that Dr. Calmeil got up and asked him whether he still persisted in thinking that epilepsy could not be feigned.

This incident not only stresses the fallacy of relying on isolated phenomena to make a diagnosis; it also accentuates the difficulties in the diagnosis of convulsive episodes. Only when one is certain of their presence has one the right to label the patient "an epileptic". Of these manifestations, one would expect the major convulsive seizure to be the easiest to recognize.

Let me then enumerate the difficulties in recognizing its occurrence. The patient's account of the seizure is usually vague, because the pre-convulsion and post-convulsion amnesias make him unaware of the changes that occur at that time. The testimony of bystanders is of doubtful worth, because the suddenness and the dramatic nature of the event "hypnotise" them into a state of inaccurate observation. Their concern is with holding down the struggling patient. The changes that the medical man has been taught to associate with this commotional event have usually passed by the time he arrives on the scene. Should he then be aware of the disturbances of higher psychical functions that persist, it would be possible to suspect that a seizure had occurred.

The need, then, to delineate more carefully this post-epileptic period is obvious. One reason for its incomplete description may be that authors have based their works on the seizures observed in institutional patients. The mental defectiveness of such patients precludes an adequate investigation of the mental state.

One may conclude that a careful evaluation of the psychological phenomena of the post-convulsion period will usually allow a clinical diagnosis to be made. Our teaching makes us sceptical that psychological findings can be so diagnostic as physical signs. I have substantiated these facts by giving "Cardiazol" to proven epileptics, and they maintain that the post-convulsion period of the induced seizure and that of the naturally occurring seizure are identical.

The one clinical group which presents diagnostic difficulties consists of those patients of poor mentality complaining of "turns" of which no adequate description can be obtained, the examination of whom at the time of the "turns" is impossible. One wishes to know whether they are hysterical, or epileptic, or both. Usually they are hysterical, and this view gains support if they do not respond or if their seizures do not lessen when anti-convulsant treatment is given.

Let me digress to state that after the "Cardiazol" convulsion, the most persistent physical sign is that the plantar reflex is extensor in type. The pupils more rapidly regain their normal reactions, often during the unconscious period. Romberg is stated to have made the following observation: "In the third stage the pupil is insusceptible when the candle light or sun is turned upon it. There is no sign so trustworthy for distinguishing between feigned and genuine epilepsy."

Perhaps, instead of harping on the clinical recognition of this condition, especially when one considers its difficulties, I should have devoted more time to the value of the electroencephalogram in elucidating these problems. The tracings of epileptics show that in the resting phase 50% are of an abnormal type. Tracings taken at the time of the seizures reveal features characteristic of each type of the epileptic manifestation. The focus for the discharge is in the pre-frontal region in major epilepsy and in the post-central region in minor epilepsy. The various types of alteration in the tracings produced in the seizures depend on their nature. In *grand mal* it is a fast rhythm, in *petit mal* it is a mixed slow and fast rhythm with spikes and waves, and in psychomotor attacks it is a slow smooth wave. Overbreathing increases the size of the waves in potentially epileptic children. The tracings taken during the "turns" will differentiate the true from the hysterical condition; but if the patient is seen at such times, this should be possible by clinical means.

One may then state that the value of the electroencephalogram in epilepsy is great; but, as in other spheres of medicine, a true perspective is gained only by the consideration of technical findings in conjunction with the clinical picture.

The Aura of Epilepsy.

An aura is said to precede about 60% of major convulsive seizures. Even then the patient's description of it is frequently vague and indefinite. In my experience this figure is too high, and with "Cardiazol" therapy an aura is even more uncommon. Its usual form is a pulsating shadow, which increases in intensity till the "blackout" occurs.

A rare but interesting aura occurred in the case of a refugee woman from the last war. It took the form of her relatives coming one by one and placing their hands on her head. Between treatments she denied being able to recall their features, and yet this vivid and terrifying experience preceded each injection.

I was more interested in seeking the reason why in the majority of cases the aura is absent or ill-defined. Several explanations can be given. It may be due to the patient's lack of intellectual ability to describe such experiences. It may be due to the rapid development of the seizure, which precludes the development of such subjective changes. It may be due to the seizure's arising in a

"silent" cerebral area. Another explanation in keeping with the findings is that the pre-convulsive amnesia "blacks out" the events of this period. Whether it does one cannot say; but investigation by narco-hypnosis throws no light on this interesting speculation.

The Problem of the Epileptic Temperament and Epileptic Degeneration.

There is considerable confusion in our text-books as to the meaning of the "epileptic temperament". Some say that it is due to specific personality traits, which make the diagnosis of epilepsy possible before seizures have occurred. Others attribute it to the results of the seizures. Others again hold that it is due to treatment, especially with bromides. Let us consider each of these statements in turn.

I have had the opportunity of studying nearly 200 non-institutional epileptics. These are patients in whom the disease has not progressed far. Even at this stage the majority show personality disorders. By far the commonest group is that in which epilepsy is associated with some degree of mental defect. If, then, there is a common "temperament" of this group, it is that of the majority, it is that of the defectives. The features of this condition are difficult to differentiate from those usually ascribed to epileptic temperament. I believe that it is the occurrence of epileptic phenomena rather than any typical features of an epileptic temperament that differentiates the epilepsies from comparable psychopathic states.

Our next problem is: "Do the convulsions produce cerebral damage resulting in a specific clinical picture, an epileptic temperament?" This question is intimately bound up with another: "Do the convulsions hasten the development of the abnormal mental features already present?"

It has already been shown that impairment of the highest intellectual faculties follows convulsions. These changes are more pronounced when the basic nervous structure is abnormal and when the seizures occur at frequent intervals. These processes are hastened by the innate tendency for abnormal organs to degenerate early. It would seem, then, that a specific picture can be produced; but unfortunately it is most likely to occur in those with gross disorder, and then the innate features dominate the clinical picture.

The last consideration is the part treatment plays in the production of the disorder. Bromism, like any chronic toxæmia, can produce permanent changes. The nature of these changes is similar to those of the acute conditions, but their development proceeds more slowly. It is usually in the most severe types of case that bromides are given, and then in large doses to control the seizures; but this fact brings in its wake further deleterious reactions in addition to those enumerated.

One may then conclude that there is no specific "epileptic temperament" present before the convulsions ensue. There is, however, a specific feature, an abnormal personality. As the disease progresses, deterioration occurs, owing to a number of causes. The clinical picture at any stage of the disease depends on the rate and severity of the superadded factors modifying the original psychopathic state.

On the Nature of Epilepsy.

The modern definition of epilepsy as a "paroxysmal cerebral dysrhythmia" finds a place in the understanding of cerebral disorders, as do abnormal rhythms in the problems associated with cardiac insufficiency. Such terms are, however, descriptive rather than an explanation of ætiological factors.

It is only in the case of a certain number of people suffering from obvious illness, such as uræmia or cerebral abscess, that the effect on the nervous system shows itself by epileptic phenomena. Is this due to the nature of the person, to the fact that 10% of the population are shown by the electroencephalogram to have potentially abnormal rhythms? Or is it related to changes in localized nerve centres, which are responsible for the production of these rhythmic changes? In these cases we have a relatively normal person, in whom a gross disorder produces the epileptic states.

The reverse picture is seen in idiopathic epilepsy. Here the constitutional factor is considerable, and this abnormality allows subliminal stimuli to bring on the convulsive episodes. Sometimes the stimulus is recognized, the commonest being emotion. Usually it is unknown, although masquerading as "toxico-metabolic" causes. The constitutional factor is reflected in the personality studies of the subjects, which reveal abnormalities out of all proportion to those seen in comparable population samples.

Between these extremes are all gradations showing the interaction between environmental and constitutional factors. As in most branches of medicine, it is the further study of constitutional factors or the diathesis that will bring light into many of these so-called idiopathic conditions.

One may say, then, that 10% of the population show certain abnormalities in nervous action. Of these, 0·2% to 0·4% have this abnormality in a sufficiently severe degree to manifest itself by epileptic phenomena and an associated personality disorder of varying grades. Just as any abnormal organ in any bodily system bears poorly the assaults of pathological processes, so, too, does the nervous structure of these people, gradually allowing the clinical picture associated with mental deterioration to cloud the already damaged canvas.

That is a broad picture. There are many details yet to fit in. Is there a convulsive centre in the pre-frontal region where the major convulsive seizure begins? If so, why have no pathological changes been noted in this region? What is the explanation that the commonest pathological lesion is in the *cornu ammonis*? Why does the seizure take its characteristic clinical form?

Medico-Legal Aspects of Epilepsy.

Time allows only a few dogmatic statements on the medico-legal aspects of epilepsy. Crime may be due to the inherent personality disorders, such as deficiency *et cetera*, or to the degenerative changes. The nature of the crime will depend on the type of this disorder.

I am more interested with crime associated with the seizure itself. In the pre-convulsion period a person may commit a crime, which is "blacked out" from his memory by the development of the amnesia. Yet one cannot for this reason say that he was of unsound mind at the time it was committed.

Any criminal action in the post-convulsion period is a more difficult proposition. I believe that a person in this stage could be persuaded to commit a crime and yet have no recollection of doing so. If he were seen by bystanders, they would all swear that he was perfectly normal; and yet our observations show that this is not so. It is necessary for us to determine whether his mental state made him unaware of the nature of his actions. I would answer: "Yes."

Another type of crime is committed by patients during the frenzies and furors that may occur in this condition.

The Relation of Other Psychological States to Epilepsy.

In the realm of popular literature, the psychological state with the widest appeal is the split personality, and although readers and authors usually regard it as a schizophrenic reaction, the descriptions seem more akin to the fuge states resulting from psychological conflict.

A recent article on compulsive and fugue states showed them to be more frequent in epileptic and affective personalities, sometimes with both trends present in the one person. In women the condition arose most frequently at the menses. In the search for a psychological interpretation of the condition, a careful review of the histories of these patients revealed an unhappy home life, frequently the absence of one parent. The wandering episodes were the outcome of a striving for the restoration of the parent-child relationship.

Without the knowledge that a convulsive seizure has occurred, the post-"Cardiazol" period is similar to the fugue state. Reliable witnesses will vouch for the normal behaviour of the subject during this interval. The comparison is even closer when one considers how this stage is usually prolonged in certain psychopathic persons, often the types described above; how only one phase of the epileptic seizure, be it aura, seizure or post-convulsion period, may dominate the form the seizure takes; and how such occurrences are most frequent in epileptics at the menses.

There is another connecting link, in the conditions often seen with inadequate dosage of "Cardiazol". They last for some time and the patient has no memory of his behaviour during that period. He is very restless, sometimes violent, and wants to be "on the move" all the time.

It is difficult to conceive that the fugue following the administration of "Cardiazol", and comparable states occurring with inadequate dosage, have no organic background, although manifestations of the fugue may be guided by psychological factors.

These facts emphasize the frequency with which psychological states may have an organic basis, although the nature of these changes is often too delicate for us to detect.

Other Commotional Conditions.

I have endeavoured to show that a definite pattern of behaviour is produced by the "Cardiazol" reaction. It is a beautiful illustration of the modification of a constant reaction by the underlying individual make-up, just as the waves of the sea playing on different earth formations take on varying forms.

It is not amiss to believe that a similar pattern occurs in other clinical states when the nervous system is subjected to acute stress. I have in mind particularly head injuries and acute toxic and infective conditions.

A psychological study of patients with head injuries has been delayed because of their customary treatment by surgeons, the majority of whom lack the patience to observe psychological phenomena. Their meagre writings already suggest that the pre-concussional and post-concussional periods follow a similar pattern. For variations in the types of amnesia they seek an explanation in the type of injury, rather than in the type of personality. Admittedly, ætiological factors contribute their share to the clinical picture; but the prime disturbance of function is independent of their nature.

It is this lack of consideration of the personality that contributes so largely to the production of certain types of post-concussive syndrome. Often a healthy man has a mild head injury, after which he complains of headache, dizziness *et cetera*. He is kept in bed, with only the thought of his condition to comfort him. As the weeks go by his symptoms persist or grow worse, and then comes the realization that perhaps he does not present a surgical problem.

So, too, in other acute nervous illnesses. In these toxæmia and local lesions tend to obscure the general disturbance of nervous function; but this can be seen if the examination proceeds with care.

Lastly, there are the chronic states, and bromism is the one mentioned earlier. In these, too, the same type of changes are induced; but the *tempo* of their development is slower. This allows the organism time to bring into play compensating mechanisms, until ultimately they fail.

The Treatment of Epilepsy.

Just as we must avoid the error of considering the motor phenomena as the essential features of the epileptic seizure, so, too, must we remember that there are many features that call for treatment besides these periodic episodes. For this reason it is easier to consider in turn the treatment of the personality disorders and the treatment of the epileptic phenomena.

The Treatment of the Personality Disorders.

Many factors have to be considered. There are the inherent abnormalities—mental defect of all grades, temperamental and emotional difficulties. On these may be engrafted changes due to the natural course of the disease and to the destructive action of the seizures, intellectual impairment, mood alteration, altered habits and the like. Each individual is his own problem.

Another group illustrates the psychological mechanism induced by the patient's attitude to his disability. The hopeless task of obtaining work, the burden of his family, the fear of injury during a seizure, frequently produce anxiety states. These anxieties must be allayed, because they precipitate seizures.

More interesting are those rationalizations built up by the paranoid types of patient.

I may mention one man whose seizures coincided with changes in the moon. When he first came to my clinic he was so insistent that the moon was responsible for his condition that I believed his prognosis hopeless. After much persuasion I managed to alter his treatment; with the alteration his seizures ceased, and with them the above views. Now his paranoid make-up drives him relentlessly to find an occupation, and it is pitiful to review at each visit the difficulties he encounters.

Another group consists of the "weather prophets". Their "turns", usually "muscle jerks", occur with stormy weather. There may indeed be an electrical basis for this occurrence.

In all these disorders the need for occupation is one of the prime necessities of treatment. Admittedly, there are many positions which such persons could not fill; yet when we consider the assistance given to other groups with disabilities—the blind, deaf and dumb—it is not surely asking too much that some organization should be set up to help these handicapped people. Such a move is worth while, because of the ready assistance one would receive from the patient and from other members of his family. It is remarkable what hardships relatives will suffer to assist any member of their family afflicted with this disorder. One sees the mother sitting up night after night to tide her imbecile child over these "dreadful" seizures.

Recently a patient asked me, somewhat ruefully, if he could take the job of a "roof-spotter" for air raids; there could be only one reply.

The Treatment of the Epileptic Temperament.

The reasons for controlling these periodic events are to avoid danger to life and damage to body that they may cause; to avoid anxiety and other mental reactions based on their possible occurrence; and to avoid their damaging effect on the nervous system.

The drugs used for this purpose, in their order of usefulness, are "Dilantin", the barbiturates and the bromides. They may be used alone or in combination. My usual practice is to commence with one capsule of "Dilantin" three times a day, with one grain of phenobarbitone at night. If this dosage is inadequate, the amounts are increased. Should toxic manifestations occur, related compounds are given alone or in other combinations.

Although the success of these drugs is attributed to their anti-convulsant action, it is not easy to assess whether they directly affect the psychological state and ill-defined psychomotor equivalents. Support for such a view would seem to come from the successful use of bromides in many psychogenic disorders. Such lessening of emotional tension removes one precipitating factor for seizures.

The Results of Treatment.

What are the results of treatment? One hopes to abolish the seizures entirely. However, it is better to have some seizures than to have a stuporose existence. In most cases the frequency is lessened.

Sometimes an alteration in the periodicity is produced. "Turns" occurring at night or at the week-ends may be

delayed to the day-time or may occur on week-days. This may be a decided liability to a working person. On the other hand, the alteration can rarely be used to advantage if the seizures cannot be abolished.

Again, the nature of the "turns" may alter. Major reactions give place to minor ones or to "effects". An aura may develop. The patient then receives warning of the impending seizure and so has time to seek safety. Motor phases may be less active and less prolonged.

The other important factors in lessening the frequency of the "turns" are attention to general health and the avoidance of mental stress. This has already been outlined in the preceding section, and again I mention occupation as a predominating need.

Certain clinical groups present difficulties in treatment. The first is that in which convulsions occur at long intervals. Not only is it extremely difficult to discover the effective anti-convulsant dosage of drug, but also the patients have an antipathy to the prolonged taking of drugs. I believe it wise to give small daily doses of "Dilantin" and "Luminal" and to increase the amounts; at the time a careful study of the periodicity shows when seizures are most likely to occur.

A second difficult group is that in which the seizures are severe and frequent with the menses. Usually local surgical measures have already been tried without effect. As yet I have found no complete solution for the problem in this group, although the above-mentioned drugs are of assistance.

The last group comprises the patients with poor mentality who have frequent severe seizures. Perhaps one is optimistic to expect results with such abnormal material.

In most cases the treatment is prolonged. Time passes before the finer adjustments to the dosages and combination of the drugs best suited to each person can be discovered. Sometimes it may take years; but the result is worth while. There can hardly be any greater joy than to free a person from the possibility of fits.

There is a great principle in therapeutics which should not be forgotten, here less than ever; it is this, that when a disease has deeply penetrated the organism, when it masters its whole substance as it were, one cannot pretend to silence its manifestations, to cure it within a short space of time. A chronic disease requires chronic treatment.

Conclusions.

In most disorders, careful observation and evaluation of symptoms and signs may allow us to make those advances which we expect to be possible only in laboratories or with the aid of various mechanical contrivances. Even in epilepsy, in which cerebral electric potentials are magnified a millionfold, laboratory methods cannot as yet portray as nicely as the clinical picture those disturbances resulting from slowly developing or diffuse pathological processes. Such methods, however, stimulate us to turn again to the clinical picture. If then we study the clinical picture we may discover new aspects, previously obscure or unnoticed, which will give us more understanding of the clinical problem confronting us.

SULPHONAMIDES IN GONORRHŒA.[1]

By V. N. B. Willis, M.B., Ch.M.,
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This paper deals with my own experiences in the treatment of gonorrhœa in males with sulphanilamide. I have not found that such treatment was a great improvement on what I may call the older methods. It certainly stopped the discharge; but recurrences were frequent, and even if there was no recurrence it was difficult to bring about a satisfactory standard of cure. Threads in the urine persisted for a long time, and it was most difficult to clear the pus from the spermatic vesicles.

Most specialists in venereal disease have now abandoned sulphanilamide completely in favour of sulphapyridine, or "M & B 693". There is no doubt that in this drug we have a most useful weapon against gonorrhœa. It rapidly stops the acute urethral discharge, and in a certain proportion of cases it undoubtedly cures the infection without any other treatment. I should like to bring to your notice, however, the fact that before "M & B 693" came into use 50% of patients were also rapidly cured with a minimum of treatment. That is to say, the infection was so mild that a few irrigations would clear it up; or (to look a little further back to the past) a few bottles of sandalwood oil would have had the same effect. Of course, today the credit for rapid cures in such cases is given to "M & B 639". This drug does reduce the acute stage of the infection from about three or four weeks to two or three days; therefore there is little time for gross damage to occur in the urethra. The mental effect on the patient is pronounced when he sees no discharge and feels no discomfort after such a short period. But it is in these two facts that the danger in the use of the drug arises. Both the patient and his medical attendant are satisfied, and nothing more is done. We know that "M & B 693" does not kill gonococci, but merely stops them from multiplying. In fact, its action on the organism is the exact opposite to that of alcohol, which causes them to increase in numbers.

Cases of gonorrhœa may be either (i) primary or fresh infections or (ii) secondary or recurrent infections. What happens in a fresh infection? I am certain that the gonococcus does not lurk, say, one-quarter of an inch inside the meatus for a week or two after the patient's exposure to infection, but that owing to a sort of "suck-back" action after ejaculation it may lodge quite a long way down the urethra. It then penetrates the mucous membrane or the ducts of some of the glands which open into the urethra. Hence it may lodge in the glands of Littré or Cowper, in the ducts of the prostate or in the spermatic vesicles. Then it begins to breed and a urethral discharge occurs. In fact, the longer the incubation period, the more likelihood is there that the infection has already involved the glands opening into the posterior portion of the urethra. In a fresh infection with an incubation period of, say, four weeks the vesicular secretion on the first day will be found to contain pus cells. In other words, the infection begins as a posterior urethritis.

This is where most of the trouble with "M & B 693" occurs. The drug stops the acute discharge, but unless the original focus in the posterior portion of the urethra is treated at the same time and kept open, the discharge will recur. Then, if more "M & B 693" is given, the patient gradually becomes immune to the drug and it becomes useless. In these so-called resistant infections, examination of slides made from the discharge will reveal almost as large a bulk of gonococci as of pus cells. In fact, I have in no other cases seen so many gonococci in a film. Naturally, in recurrent infections there is a ready-made focus, usually in the spermatic vesicles, and all that "M & B 693" does is to control the acute relapse. It certainly does not clear up the focus.

At the present time there are two methods of using "M & B 693" in the treatment of gonorrhœa. The first is to use massive doses in a short period—44 tablets in forty-eight hours. The patients, of course, must be kept in bed and given only a milk diet. The second method is to give four tablets per day for one week. I have not tried the first method, but I am satisfied with the second procedure. I think that the tablets should be taken at once and that daily irrigations are of considerable value. The course of treatment may be repeated if necessary after an interval of one week. When the discharge has stopped, proper tests of cure must be carried out to make certain that no

[1] Read at a revision course of the Queensland Post-Graduate Committee on November 8, 1941.

organisms have been left lurking beneath the mucous membrane or in one of the secretory glands. Both methods of treatment stop the acute discharge, but neither method will cure an infection which begins posteriorly and involves the vesicles. These must be treated at the same time until free drainage is established. In relapse due to an old infection proper massage must be carried out, as nearly all relapses are due to dormant foci in the vesicles.

I must repeat that "M & B 693" will rapidly stop an acute urethral discharge, but that if this discharge comes from a focus in the posterior portion of the urethra, the focus must be treated or further relapses will occur.

Since the introduction of "M & B 693" in the treatment of male gonorrhœa, I have noticed that:

1. The incubation period for gonorrhœa has become much longer. In one undoubted case the period was actually seven weeks; but periods of three or four weeks are now quite common. In my opinion this is due to treatment of the source of infection by the drug; the infecting woman thinks she is cured, and the rate of growth of the lurking gonococcus when it is introduced into a new medium has been diminished.

2. Cases of acute gonorrhœa are not so common as they used to be; although just as many cases of acute urethritis occur as formerly, most of them are found to be due to Gram-negative diplobacilli. Clinically the condition of patients with such infections resembles that of patients suffering from gonorrhœa, and I have proof that they are just as infective. I think that the gonococcus has taken on a new form. At present attempts are being made to cultivate these organisms.

3. Gonococci are present in the mildest cases of urethritis. There is not much pus formation and it is difficult to obtain enough secretion for examination.

4. The incidence of complications such as epididymitis has lessened considerably.

5. Strictures in the anterior portion of the urethra are becoming more common, because the organisms beneath the mucous membrane in mild cases lie dormant and cause more rapid infiltration than in the past. Hence the necessity for the use of sounds.

Conclusion.

I have tried to show you (i) that "M & B 693" stops the urethral discharge at any stage, but does not cure the infection except in early primary cases; (ii) that the administration of large doses of the drug serves no useful purpose; (iii) that the drug should never be given over long periods, because it then simply makes the patient's condition worse. In "M & B 693" we have a most useful weapon in an attack against gonorrhœal infections. None the less, I feel that in the years ahead there will be no lessening of the morbidity due to these diseases, and I see no present indications, after the drug has been in use for two years, of any lessening of the number of urethral infections, although they are certainly much less severe and are usually found to be due to organisms that are not typical gonococci. The danger that patients with these milder infections will spread the disease is much greater.

When we treat a patient suffering from gonorrhœa, we should aim (i) at rendering him non-infective and (ii) at repairing the damage caused by the infection. "M & B 693" is of help only in the first of these objectives. As far as the second is concerned, much trouble would be avoided if the public and the medical profession could be educated to demand that clearness of the urine should be the sign of complete repair.

In conclusion, let me state that to tell a patient, without bothering to put him through any tests whatever, that he is cured of his gonorrhœa because the discharge has stopped after he has taken 25 tablets of "M & B 693", is about equivalent to telling a syphilitic patient that his infection is cured as soon as his primary sore has healed.

Reports of Cases.

MUMPS MENINGO-ENCEPHALITIS.

By P. W. GILL,

Captain, Australian Army Medical Corps, Medical Officer, Military Ward, Base Hospital, Tamworth.

SEVERAL cases of mumps meningo-encephalitis among soldiers were reported by A. W. Frankland in the *British Medical Journal* of July 12, 1941, at page 48. He observed that parotitis and orchitis were not always present; that when this was so the clinical picture was sometimes indistinguishable from cerebro-spinal fever; that the number of cells in the cerebro-spinal fluid ranged from 230 to 1,078 per cubic millimetre, lymphocytes generally predominating; that attempts at culture were sterile; and that patients who did not receive sulphapyridine recovered more quickly than those who did. The biochemical findings in the cerebro-spinal fluid were not recorded.

The cerebro-spinal fluid findings in this condition are detailed further by G. A. Harrison ("Chemical Methods in Clinical Medicine", second edition, 1939), who summarizes them as follows: protein content, 30 to 130 milligrammes per 100 millilitres; chloride content, 670 to 750 milligrammes per 100 millilitres; glucose content, 70 to 115 milligrammes per 100 millilitres; cells (mainly lymphocytes), 300 to 3,000 per cubic millimetre; attempted culture, sterile.

In the light of these observations the following two cases are reported.

Case I.

A soldier, aged nineteen years, was admitted to hospital on December 22, 1941. Headache had been present for four days, and vomiting was now occurring. He was cooperative and his mental condition was normal. Slight neck rigidity was present. The temperature was 102° F. and the pulse rate 110 per minute. There was no history of contact with meningitis, but some of his hut mates had mumps and some rubella. He, however, did not develop parotitis or orchitis. At lumbar puncture the cerebro-spinal fluid was faintly opalescent and under increased pressure. It contained 700 cells per cubic millimetre, 55% being neutrophile cells and 45% lymphocytes. Attempted culture was sterile. The fluid was not biochemically examined. The patient was given sulphapyridine by mouth. The initial dose was five grammes, followed by two grammes every four hours for twenty-four hours, and then one gramme every four hours. Although his condition improved after lumbar puncture and continued to do so, his temperature remained elevated until December 26 (the fifth day of treatment), and thereafter it was normal. Nausea and anorexia were troublesome while chemotherapy lasted. On December 29 he developed a rash and posterior cervical adenitis. This was thought to be rubella, but may have been a toxic effect of the sulphapyridine treatment, which was accordingly suspended. After that he felt much better and convalescence was uneventful.

Case II.

A soldier, aged twenty-five years, was admitted to hospital on January 9, 1942, complaining of severe headache of twenty-four hours' duration and of repeated vomiting. He was drowsy and apathetic. Neck rigidity was pronounced and Kernig's sign was easily elicited. His temperature was 99·8° F. and his pulse rate 60 per minute. Lumbar puncture yielded clear cerebro-spinal fluid under normal pressure. It contained 350 cells per cubic millimetre, 10% being neutrophile cells and 90% lymphocytes. The protein content was 120 milligrammes per 100 millilitres and the chloride content 740 milligrammes per 100 millilitres. The glucose content was not estimated. He felt much better after lumbar puncture, but for safety's sake was given sulphapyridine by mouth, the dose being six grammes followed by one gramme every four hours. The treatment was discontinued on the following day, when attempted culture from the cerebro-spinal fluid was reported to be sterile. The total dose given was 12 grammes. On January 11 his temperature rose to 101° F., but he felt well and headache was not troublesome. On the next day (the fifth day of illness) he developed swelling of the right parotid gland, and thereafter his illness was a straightforward attack of mumps. His temperature remained between 100° and 101° F. until January 15, when it fell to normal. Convalescence was uneventful.

Comment.

Case I was somewhat indefinite, as the cerebro-spinal picture resembled in some respects that of a mild type of meningococcal infection (D. Brinton, "Cerebrospinal Fever", first edition, 1941, page 70). Chemotherapy had been in full swing for several days before meningo-encephalitis of mumps was even thought of, the thought being prompted by the high proportion of lymphocytes and failure of sulphapyridine to influence the temperature. Case II needs no comment.

CONGENITAL ATRESIA OF THE ŒSOPHAGUS.

By GEOFFREY HALL, M.B., B.S. (Melbourne),
Fremantle Hospital,

THE following case of congenital atresia of the œsophagus is, I feel, of sufficient rarity to warrant publication.

Clinical Record.

The baby was first seen at the age of three days, when the following history was given. The infant would swallow, but not suck. The fluids swallowed were almost immediately regurgitated in a frothy condition. There was also a constant frothy nasal discharge. The child was subject to mild cyanotic attacks, and these were precipitated by attempted feeding. It was thought that no fluids at all had been retained during the three days of life. The bowels had not acted. The mother's confinement had been uncomplicated, and at birth the baby had weighed seven pounds and appeared perfectly healthy.

On examination the baby showed no signs of pulmonary embarrassment, cyanosis was not present, and the heart, chest and abdomen appeared normal. Sterile water was then administered by mouth and the effect was noted. The water was swallowed, and no abnormality was noted until ten to twenty seconds after its administration, when the child consistently spluttered, coughed up frothy fluid and became cyanosed. All these symptoms passed off within a few seconds. An attempt was then made to pass a small catheter into the stomach, but this consistently blocked at what was considered to be the upper part of the œsophagus. On this evidence the diagnosis of congenital atresia of the œsophagus was suggested.

On the fourth day, however, certain changes took place. Two small motions of meconium were passed. The abdomen became a little distended, especially in the epigastrium. The cyanosis became more pronounced and was present between feedings. The child's temperature rose to 103° F. and inspiratory moist sounds were noted at the base of the right lung. Moreover, the sister in charge reported that the child was certainly retaining some fluid; it had in fact taken one ounce of milk without any regurgitation. On this additional evidence the diagnosis of œsophagospasm and secondary pneumonia was put forward. The child was forthwith treated with belladonna, "Sodium Dagenan" and saline solution given intraperitoneally.

On the fifth day the child was very ill. The X-ray plant was available, but it was considered inadvisable to make a barium bolus examination of such a sick patient. However, an X-ray examination of the thorax was made with the portable plant, and the only abnormality revealed was a large quantity of gas in the region of the stomach. The child died on the sixth day.

Post-Mortem Examination.

Post-mortem examination revealed that the cause of death was congenital atresia of the œsophagus and secondary pneumonia of the lower lobes of both lungs. The upper end of the œsophagus ended blindly just below the cricoid cartilage; the lower end opened by a small longitudinal slit into the posterior wall of the trachea, half an inch further down. A probe was easily passed from the trachea through this slit down the œsophagus into the stomach. From this it can be seen that the only method of entry of food into the stomach was through the *rima glottidis*, down the trachea, through the longitudinal slit and so into the lower segment of the œsophagus. Hence any fluid retained must have either followed this route or entered the lungs. No signs of milk were found in either the lungs or the stomach. The stomach contained a large quantity of air and some thick brown fluid. In the lungs extensive pneumonic consolidation in the stage of red hepatization was found. No other abnormality was detected.

Comment.

The following points are of interest in this case.

1. A child with complete atresia of the œsophagus certainly retained some fluid taken by mouth. It is important to remember such a possibility in the diagnosis of the condition.

2. An attempt to pass a tube into the stomach forms a valuable point in the diagnosis. A barium bolus examination was not made in this case because the X-ray plant was not available when the infant was in a fit condition to undergo the test.

3. The presence of air in the region of the stomach in the X-ray film and the absence of air elsewhere in the abdomen were confirmatory evidence of a communication between the lungs and the stomach. Had the air been due to fermentation, one would have expected to find it also in the intestines. It is said that this communication can be diagnosed clinically by the filling of the stomach with air during inspiration. This sign was looked for but not detected in this case.

Conclusion.

A case of the rare condition of congenital atresia of the œsophagus is described. The infant died in the typical way from pneumonia.

Acknowledgements.

My thanks are due to Dr. F. H. U. Baker and Dr. C. Christie for allowing me to publish this case.

Reviews.

MASSAGE AND MEDICAL GYMNASTICS.

THE presentation of the practical aspect of massage and remedial exercises is very difficult by means of the written word, but Mary V. Lace has dealt with the subject in a masterly manner in her second edition of "Massage and Medical Gymnastics".[1] Written essentially for students, the book is divided into two parts; the first deals with massage, but only thirty pages are devoted to this subject, while the second part is devoted entirely to medical gymnastics. The subject matter is well presented under numerous headings with an effective orderliness, while the numerous illustrations help the understanding of the text considerably.

The author makes little claim for originality and points out in the preface that most of the exercises in the second part of the book are derived from Dr. Arvedson's classical work on the subject.

Particular stress is laid on starting positions for the different exercises, and, after an explanation of the latter, the muscles and the joints moved are enumerated; then there follows an account of the effects and uses of the exercises.

There is an index which appears to be complete. For the student of massage and medical gymnastics the book should prove very helpful.

REFERRED PAIN.

IN a short paper entitled "Referred Pain: A New Hypothesis" G. W. Theobald deals with a good deal of the work which has already been published and with some new considerations advanced by himself.[2] These considerations lead the author to believe that the areas of hyperirritability are spread throughout the apperceptive centres of the nervous system and cannot be considered to be localized to the spinal segment. He adduces considerable evidence for this view, which he calls a "new hypothesis". Many people with the evidence which the author has to bring might have been tempted to call it a "law". This restraint in claim for magnitude exemplifies the author's sound judgement throughout the paper.

[1] "Massage and Medical Gymnastics", by M. V. Lace, with a foreword by J. Mennell, M.A., M.D., B.C.; Second Edition; 1941. London: J. and A. Churchill Limited. Demy 8vo, pp. 250, with 120 illustrations. Price: 12s. 6d. net.

[2] "Referred Pain: A New Hypothesis", by G. W. Theobald; 1941. Colombo: The Times of Ceylon Company Limited. Crown 4to. Price: 2s. 6d.

The Medical Journal of Australia

SATURDAY, APRIL 11, 1942.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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MEDICAL CERTIFICATES AND THEIR CREDIBILITY.

It was reported in the Melbourne *Age* of February 7, 1942, that in the Federal Arbitration Court on the previous day, before Judge O'Mara, Mr. C. H. Grant, representing the Victorian Chamber of Manufactures, declared that he doubted the genuineness of medical certificates produced by some workers in war industry as an excuse for their absence over Christmas holidays. Mr. Grant was further reported as having alleged that of six certificates presented to one Victorian firm, three were from one doctor. The newspaper account went on to state that when in answer to a question Mr. Grant said that the employees concerned worked long shifts, some of which were of twelve hours' duration, Judge O'Mara expressed the view that doctors could quite honestly have given the certificates. Mr. Grant also said that employers found it difficult to accept these certificates, and were unable to distinguish between the genuine and the false. Since this report was published in *The Age* somewhat similar statements have been made by insurance company officials and others connected with industry. Mr. J. O'Toole, of the Boiler Makers' Society of Australia, is reported in *The Age* to have declared that the statement made by Mr. Grant was a reflection on the medical profession, and this he was not prepared to accept. Clearly statements of the kind mentioned cannot be allowed to pass any longer unnoticed. While it is possible that among the members of the medical profession in the Commonwealth there may be some who would deliberately give a misleading certificate and by so doing sell their birthright for "a mess of pottage", we believe that such members, if they do exist, are very few in number. When misleading certificates are given—and unfortunately the fact cannot be denied—the act is nearly always the result of carelessness. The present occasion is therefore an urgent appeal to all doctors in Australia lest by want of thought they not only bring discredit on themselves and dishonour on their profession, but jeopardize the safety of their country, which stands in urgent need at present of all that those engaged in industry can give it.

Before any discussion on certification in general is undertaken reference must be made to the opinion expressed by Judge O'Mara that the certificates to which Mr. Grant took exception could have been given quite honestly by medical practitioners. The learned judge is evidently not like some political leaders and industrial employers who by their actions seem to deny that there is such a thing as industrial fatigue, or who, if they know it to exist, prefer constantly open factories producing less than the maximum output and staffed by fatigued workers, to factories, not necessarily always open, but staffed by energetic men and women working for fewer hours and producing the greatest possible output. There is no need to traverse all the arguments about industrial fatigue put forward in these columns from time to time and advanced with such cogency in an address by Dr. H. M. L. Murray published in this journal last October—the facts are proved beyond any doubt. At the same time, it is not for this journal to question the wisdom of the Minister who decreed that factories should not close between Christmas and the New Year; there were obvious reasons why the Minister made his decision. The trouble was that many workers after toiling in twelve-hour shifts for many months showed signs of fatigue, and there is no doubt that a large percentage would have been entitled to medical certificates on that account had they sought them. The practical lesson which the medical practitioner should gather from this is that when he gives a certificate because a worker shows signs of industrial fatigue, he should state the diagnosis without equivocation and if possible add some explanatory remarks to show suspicious employers and insurance companies that the diagnosis is deliberately made and will if necessary be defended. If, apart from the workers themselves, medical hygienists were not the only, or practically the only, people who recognized industrial fatigue as a real entity, the explanatory remarks would not be necessary.

But some reference to medical certification in general is necessary. The reported episode in the Federal Arbitration Court is only one of the reasons for the present discussion; moreover, though it is probable that many of the cases that brought about the reference in court arose from industrial fatigue, it is very unlikely that they were all of this nature. Further, the present is a time of intense and increasing industrial activity, and much more watchful care is needed than in normal times. The work of a medical practitioner is not completed when he has made a diagnosis and given certain treatment, and even when his patient has recovered. The patient may be the employee of a business firm or other body, he may belong to a friendly society lodge or other cooperative society, he may be insured under the *Workers' Compensation Act*. If so, the medical attendant will in all probability be asked to give the patient a certificate setting out the nature of his illness or injury and to state how long he is likely to be incapacitated. Before the patient goes back to work the medical attendant will also be asked to certify that recovery is complete and that the patient is fit to carry out the usual duties of his occupation. In all these certificates the doctor must

distinguish sharply between facts and opinions. It is a fact, for example, that a certain patient has broken his leg. The medical attendant may have to rely on hearsay evidence as to whether the bone was broken during the course of the patient's usual employment. The statement as to how long the patient will be away from work and a declaration that recovery is complete and the patient fit to resume work, are only opinions. Never must hearsay evidence be stated as fact—the words "I am informed that . . .", "I understand that . . .", "The appearances are consistent with the statement that . . ." or some similar expression must be used. Sometimes the medical attendant may be in doubt as to whether the patient should be ordered to rest. In these circumstances the majority of experienced practitioners will give the patient the benefit of the doubt, since they would prefer to make him rest unnecessarily rather than allow him to run the risk of damaging his health by a continuance of work when he should be at rest. Sometimes it may be advisable for the medical attendant to tell the employer, friendly society secretary or insurance company manager that he is in doubt, and therefore proposes to give the patient the benefit of the doubt. Such a course will give evidence of the doctor's *bona fides* and make for mutual confidence. We can thus see that in general medical certificates are a reflex both of a practitioner's knowledge and of his integrity. They may sometimes also reflect the practitioner's ingenuity, for malingerers often try to secure certificates to which they are not entitled. To unmask a malingerer may be extraordinarily difficult, but is generally possible. In any case, a practitioner whose certificates are known to be beyond reproach will in all probability not be blamed if an occasional malingerer is successful in deception.

So far we have presumed in this discussion that the practitioner's intentions are good, that his certificates express, as indeed they should, an impartial view (in nine cases out of ten certificates contain an expression of opinion) and that nothing would be further from his mind than a wish to mislead either the patient or his employer. Here, however, we must draw attention, as we have done on previous occasions, to two reprehensible practices which in all probability are adopted either from sheer good nature or from laziness. One of these practices is the post-dating of a certificate and the other is the issuing of a certificate by a practitioner who has not examined or perhaps even seen the patient. A moment's thought will show that the practitioner who does either of these things is most blameworthy. He is asking for trouble and may easily find himself in the unenviable position of having to face a charge that he has been wilfully misleading. In other words, he will have no cause for complaint if he is classed among the few who may or may not exist and who set out to be deceptive and to sell themselves for gain. Persons of this sort would be beneath contempt, and it is to them that the warning notice of the General Medical Council of Great Britain would apply. This notice runs as follows:

> Any registered medical practitioner who shall be shown to have signed or given under his name and authority any such certificate, notification, report or document of a kindred character which is untrue, misleading or improper, whether relating to the several matters above specified or otherwise, is liable to have his name erased from the medical register.

The conclusion of the matter is that the whole question of certification must be put on the highest possible ethical plane; it must be regarded as a matter which concerns vitally the honour of the individual practitioner and of the whole profession. No man likes to be called a liar. The doctor who is careless with certificates must not be surprised if people think that he is a liar. If he is proved to be guilty of deliberate lying he should be dealt with according to the warning notice of the General Medical Council. The profession will not mourn the loss of such a man.

Current Comment.

RHEUMATIC INFECTION IN CHILDHOOD.

CLINICS devoted to the care of children suffering from rheumatic heart disease have been instituted long enough in England and the United States of America to permit reliable statistical conclusions to be drawn as to prognosis and trends in the nature of rheumatic infection. It is now possible to offer proof of many truths long suspected by clinicians.

Rachel Ash, of the Children's Hospital, Philadelphia, has recently reviewed fifteen years' experience in the rheumatic clinic of that hospital, and reports the results of a follow-up of the patients who attended during this period for primary manifestations of acute rheumatism. In 1939 no less than 527 children, representing 90·4% of the personnel of the clinic, were traced. Others had been traced for from three to nine years previously. In all, the course of the disease in 553 children was known over a minimum period of three years and over an average period of 9·6 years. Among these, signs of heart disease had been observed at one time or other in 414 (71%), but because of regression of such signs in 59 children a diagnosis of organic valvular damage had been made in 64·2%. One hundred and forty-six (26·4%) had died of rheumatic infection only, thirteen (2·3%) of superimposed bacterial endocarditis, seven (1·3%) of an associated infection, and six (1·1%) of unknown causes. At the final examination of the 398 survivors, 208 (52·3%) presented signs of organic valvular disease, in 48 (12%) doubtful murmurs were audible, while 142 (35·7%) showed no signs of heart damage. Twenty-two were ill with acute recurrences, and ten were cardiac invalids. These figures agree with those of Carey Coombs, White and many other observers. They present the rheumatic infection as the greatest killer in the childhood years after infancy. They show that the chances of absolute escape after the rheumatic attack in childhood are only about one in three.

We are greatful, however, for any indication which may enable us to detect the lucky one in three, and Rachel Ash offers at least one useful consideration. She analysed her figures further to determine what influence, if any, the type of onset has upon the prognosis. Forty-seven (8%) of her series showed evidence of acute carditis on first presenting themselves. These were young children averaging 5·7 years of age. A third of them (30%) had an illness which ran a rapidly fatal course, and two-thirds (65%) were dead at the end of ten years. Half of the whole group, however, began their sickness with the usual attack of polyarthritis, that is, migratory joint pains with or without swelling. The average age of this class was 6·6 years. Only 59% of these children showed heart disease at the onset. One-tenth were dead at the end of the first year, and a third at the end of ten years. Three-quarters of this group showed heart disease after ten years. Those who had definite heart damage in the first polyarticular attack showed the highest mortality, while of those in whom only "potential or possible" heart disease was noted, only one-quarter ultimately became definite cardiac sufferers.

Chorea attacked 116 of the older children, and was a relatively benign manifestation; one-fifth had heart damage after one year, and 41% after ten years, while 16% had died. Fifty-three children suffered from myalgia and joint pains insufficient to force them to bed, but none the less associated with an increased sedimentation rate, leucocytosis and slight fever. They escaped not much less than those with frank polyarthritis, as heart disease was noted in 47% at the end of the first year and in 58% at the end of ten years.

A satisfying and hopeful aspect of this investigation followed. Rachel Ash was able to show that in Philadelphia at least there has been a progressive lessening of the severity of rheumatic infection, whatever the mode of onset. There has been no consistent decrease in the number of recurrences. There had been a racial distribution of the city's population with an influx of coloured children. This, if anything, should have increased the severity of the infection. The policy of the hospital regarding admissions had not changed. Ash modestly concludes there has been a spontaneous change in the character of the disease. This decreased mortality rate has been described by Hedley as general throughout the United States of America, and it would be interesting to know whether the same decrease has occurred in Australia. If not, it is because we have not realized the supreme importance of an organization whereby children are retained in hospital until the last evidence of rheumatic activity, particularly in the primary infection, has quite disappeared before transfer of the patient to a convalescent home. The value of the erythrocyte sedimentation test in this connexion should be reemphasized. This prolonged care in hospital wards is necessary, regardless of the current opinion as to whether the heart has escaped damage or not. In Kensington, London, where occupation of hospital beds by rheumatic children for a minimum period of six months was provided, the incidence of heart disease among school children fell from 2% in 1926 to 0·8% in 1936. Convalescent homes for cardiac children should be reserved for those who have need of good environmental conditions to give them a fresh start, who are convalescent from first infections, when this infection is, as far as can possibly be determined, inactive. Such a convalescent home is useless if simply intended to accelerate the departure of rheumatic children from the wards of a general hospital. With any degree of active infection, absolute bed rest for six months is essential, and any better trend in the sequelæ of rheumatic infection can be attributed mainly to a more widespread obedience to this law.

"CHRONIC CARDIAC COMPRESSION."

ONE of the chief of the so-called "compression disorders" of the heart is Pick's disease or constrictive pericarditis, so well publicized by Paul D. White in his St. Cyre's Lecture five years ago. In this condition the heart is encased in a fibro-calcareous carapace which prevents dilatation in the presence of cardiac failure. Failure therefore expresses itself in a raised venous pressure, hepatic enlargement and ascites. A respectable number of surgical cures are on record, produced by peeling off the layers of encircling fibrous tissue, especially about the entrances of the *venæ cavæ*. Certain questions, however, have remained to be answered. Is the compressed heart of actually normal size or smaller or larger? Is the pull upon adhesions a factor in producing failure? Should the pectoral muscle and bony precordium be removed in addition to the fibrous cloak as a number of experienced cardiothoracic surgeons have advocated? In addition, phrenic avulsion has been recommended to release the pull of diaphragmatic adhesions. Adhesions have generally been held to produce cardiac hypertrophy.

Experimental studies wherein chlorinated soda has been injected into the pericardial sacs of animals to induce adhesions suggested that the Pick syndrome could be produced without any cardiac adhesions. Another significant observation was that when the Pick syndrome developed, the heart became smaller than normal, but returned to normal size on excision of the pericardial scar. X-ray shadows include both the heart and the thick scar, and are therefore unreliable. Joseph Roberts and Claude Beck, of Cleveland, have studied the question by actual measurement of the diameter of the cardiac muscle fibres in five affected human hearts.[1] An exact correlation has already been known to exist between the diameter of cardiac fibres and the size of the heart. The patients from whom the specimens came had all suffered from extensive pericardiac adhesions and had died from cardiac failure subsequent to the removal of the scar. One hundred measurements were made in each case from sections of both right and left ventricles. The average mean fibre diameter in a normal heart is 13·9 microns. The mean fibre diameters in the hearts studied were 8·1, 8·9, 10·1, 11·2 and 11·2 microns, averaging 9·9 microns. The differences are of course obvious even to qualitative histological inspection, representing a reduction in fibre diameter of 27·2% of the normal value. Similar results followed artificial compression of dogs' hearts by linen tape, which was gradually tightened, in a series of experiments extending over several months and terminating in death by cardiac failure. In both the human and dog hearts the fibres were uniform and circular in cross-section, contrasting with the variable fibre atrophy seen in wasting diseases (brown atrophy).

This, then, is the answer to the question as to whether pericardial adhesions produce cardiac hypertrophy. Adhesions do not produce hypertrophy in the absence of valvular disease or hypertension, in either laboratory animals or human beings. No indication would thus exist for removal of the bony precordium or for paralysis of the diaphragm. Beck has not done more than excise the compression scar from the heart of 37 patients. Of these patients, 25 may be regarded as cured. The condition of two additional patients was improved and seven died; three of these seven had active tuberculosis of the pleuræ and mediastinum. It may be concluded, then, that the heart muscle, if not invaded by the fibrous tissue, had merely undergone disuse atrophy, and immediately the fibrous gyves were released the muscle began to regain its normal functional and structural status, as in every instance the circulation showed immediate improvement, although time naturally elapsed before it could make a full recovery.

The final and conclusive chapter in this investigation would be the examination of a biopsy specimen of cardiac muscle after successful surgery. This should not be difficult in the case of the dog whose heart was artificially constricted. It was formerly generally taught that some of the largest "bovine" hearts in man were encountered in the presence of long-standing pericardial adhesions, but subsequent analysis of many specimens has revealed the coincidence of valve disease or rheumatic myocarditis or the former existence of high arterial tension. It is encouraging to those faced with the great responsibility of deciding upon or conducting such a difficult and dangerous operation to have the assurance of a pioneer like Beck, that in most cases there is every likelihood that the muscle will restore itself almost to normal once it is given a chance, by careful, if radical, surgery.

[1] *American Heart Journal*, October, 1941.

N. HAMILTON FAIRLEY, F.R.S.

ANOTHER Australian graduate, Dr. Neil Hamilton Fairley, has been elected to the Fellowship of the Royal Society. Dr. Fairley was for some years engaged in research at the Walter and Eliza Hall Institute, Melbourne. His research, which has earned him a world-wide reputation, has extended into biology, pathology and tropical medicine; it is known to many Australian practitioners, who will join with us in offering him warm congratulations. The honour will be more appreciated from the fact that Dr. Fairley is consultant physician to the Australian Imperial Force.

[1] *American Heart Journal*, September, 1941.

Abstracts from Medical Literature.

MEDICINE.

Diabetes Mellitus.

W. C. STADIE (*Annals of Internal Medicine*, November, 1941) discusses fat metabolism in *diabetes mellitus*. There are two theories of diabetes, one that the tissues are unable to acidize sufficient carbohydrate so that ingested carbohydrate and that derived from protein are excreted, the other that the liver produces an excess of glucose from fatty acids as well as from protein in diabetes. In either case fat metabolism is important. There are three theories on the last-mentioned subject: (i) that fatty acids are oxidized to ketone bodies *plus* acetic acid; (ii) that fatty acids are oxidized to ketone bodies *plus* glucose; and (iii) that fatty acids are oxidized to ketone bodies only. The last is a new theory in support of which evidence is produced. The livers of cats with fully developed ketosis were examined. No acetic acid was found and no evidence that fatty acid was converted to glucose in the liver. The diabetic who is unable to utilize carbohydrates fully must fall back on fat for his energy requirements. This is partly met by fat oxidation in the muscles and partly by oxidation of fats in the liver to ketone bodies only which are used for energy. However, when the demand for fat Calories exceeds 2·5 grammes of fat per kilogram per day, ketone bodies in excess of needs are formed by the liver and excreted in the urine. If this is unchecked ketosis and coma follow.

Essential Hypertension.

W. RAAB (*Annals of Internal Medicine*, May, 1941) has reviewed the literature and discussed the causation of essential hypertension. It is well known that cerebral ischæmia may give rise to hypertension, and arteriosclerotic changes which suggest local ischæmia have been found in the medulla of 50% of hypertensives. Arteriosclerosis in the brain can apparently cause hypertension without vascular changes in the kidneys; but there is some doubt whether analogous vascular changes in the kidneys can be excluded in these cases. Artificial renal hypertension is abolished by pithing, which suggests some central vasomotor influence. The suprarenal glands and the pituitary are not immediately responsible for essential hypertension. Hypertonin from the serum of hypertensives causes hypertension only in the presence of adrenals. Various sterols, such as desoxycorticosterone, progesterone, œstradiol *et cetera*, have caused hypertension in rats. These various facts suggest that essential hypertension may be due to central brain causes, such as ischæmia or arteriosclerosis, to renal, adrenal and pituitary disorder, and to certain sterols, including the synthetic sex hormones.

Cold-Hæmagglutination in Raynaud's Syndrome.

T. H. C. BENIANS AND W. R. FEASBY (*The Lancet*, October 25, 1941) found on investigation of the blood of two women suffering from Raynaud's syndrome that autohæmagglutination occurred when the blood was cooled. This was due to powerful agglutinins which became operative when cooling occurred. The authors suggest that the clumping of red corpuscles within the vessels played a part in the production of the Raynaud's syndrome and that sensitivity to cold played a part in the production of both.

Glandular Fever with Jaundice.

L. MARTIN (*The Lancet*, October 25, 1941) presents reports of two cases of glandular fever accompanied by jaundice. The jaundice was supposed to be due to enlargement of lymph glands related to the bile passages.

The Hypertensions.

IN discussing the various ætiological factors in the hypertensions, A. Rae Gilchrist (*Edinburgh Medical Journal*, November, 1941) remarks first on the estimation of blood pressure. He makes a plea for preliminary readings by palpation of the brachial artery as a check on the more accurate auscultatory findings; it is emphasized that the unaided fingers are incapable of estimating blood pressure. The factors combining to maintain the pressure in the arterial system are the heart, the great vessels, the arterioles and capillaries and veins. The types of systolic hypertension associated with a normal or low diastolic level are the bradycardia of complete heart block, aortic arteriosclerosis, aortic regurgitation, patency of the *ductus arteriosus* and arterio-venous fistula. Systolic hypertension is a compensatory mechanism and an innocent manifestation of vascular disease, provided it is not complicated by a rise in the diastolic level. In health there exists the well-known 1:2:3 relationship between pulse, diastolic and systolic pressures. There is an attempt to maintain these ratios when peripheral vasoconstrictor tone is increased, so that there is a rise in systolic pressure corresponding to the diastolic increase. With a diastolic pressure anywhere between 100 and 160 the individual may be remarkably free of symptoms provided the systolic pressure is in suitable proportion. The diastolic figure multiplied by 1·7 should equal the systolic level for the two pressure levels to be spoken of as balanced. Converging pressures are an early indication of a failing left ventricle, the systolic level declining progressively while the diastolic is maintained. In diverging hypertension, arteriosclerosis in the aorta is the commonest cause in elderly patients, whilst hyperthyreoidism and nervous excitability are the factors to be looked for in younger individuals. The regulation of arteriolar resistance is partly nervous and partly hormonal. The presence of a pressor substance in the blood stream following partial renal ischæmia is now recognized, and this acts not through the vasomotor centre, but directly on the walls of the peripheral vessels or through the intermediary of the endocrine glands. From the results observed it would be reasonable to assume that the inherent muscular tone of the peripheral vessels represents a balance struck between two opposing influences—a pressor substance produced in excess perhaps by the ischæmic kidney on the one hand, and a depressor substance by the healthy kidney on the other. The pressor substance renin elicits vasoconstriction only when allowed to interact with a pseudoglobulin fraction of the blood-renin activator. The product of this reaction has been separated and variously named angiotonin, hypertensin, ischemin. A renal antipressor substance has also been isolated and impressive results have been obtained by its use in severe cases of malignant hypertension in man. This substance is still difficult to obtain and is very costly.

Carbon Disulphide Absorption.

F. H. LEWEY (*Annals of Internal Medicine*, November, 1941) discuss the signs and symptoms of chronic industrial carbon disulphide absorption in the viscose rayon industry. One hundred and twenty workers were examined. All these workers were still fully occupied and were not complaining of ill health until directly questioned. Disorders of the central nervous system were noted in many workers. Headache, sleeplessness, numbness of arms and legs, tremors, nystagmus, ataxia, diminished pupil reactions and pains in the legs were reported. Insomnia, irritability, Parkinson's syndrome, poor exercise tolerance, contraction of visual fields, blurring of optic disks and other eye signs were frequent. The serum cholesterol content averaged 178·28 milligrammes *per centum*. Sore red tongue and polyneuritis were found in 8% of subjects examined. Thiamin excretion was low. Carbon disulphide was found in the blood and urine of all workers. Carbon disulphide is eliminated from the blood within four hours and from the urine in a day. The author suggests that there may be liver damage depleting the stock of thiamin. Furthermore, it is stated that one-fourth of the workers had symptoms and signs of cardio-vascular disease before their time. The author suggests a diet rich in vitamin B_1 and measures to prevent chronic absorption of carbon disulphide.

Carcinoma of the Pancreas.

J. EDWARD BERK (*Archives of Internal Medicine*, September, 1941) reports the result of a study which was undertaken to establish, first, the current leading impressions of the diagnostic criteria of carcinoma of the pancreas, and second, the outstanding manifestations of the disease. A poll was taken among 120 recent medical graduates, representing 34 medical schools, on the question of their impressions of the outstanding diagnostic criterion of carcinoma of the pancreas, and jaundice was selected by 92·5% as the commonest and outstanding feature of the disease; and nearly half of this number specified further that the jaundice was painless. Only 2·5% thought that pain ranked as the predominant characteristic. An analysis of 34 cases of primary carcinoma of the pancreas, proved by laparotomy or necropsy, showed that pain occurred earlier and more frequently than jaundice and was the outstanding symptom. Painless jaundice was found to occur in the series with relative infrequency. The author states that some derangement of carbohydrate metabolism, abnormalities in radiographs, and elevated concentrations of pancreatic enzymes in the blood are findings of invaluable aid in the diagnosis of carcinoma of the pancreas. The following general features of the incidence of the disease were noted from a combined analysis of his 34 cases and those already reported in the literature. The average age of patients was 56·4 years; men numbered 71·3% of the patients, with a ratio of men to women of 2·4 to 1; white persons numbered 71·4%, with a ratio of white persons to Negroes of

2·5 to 1; the average duration of symptoms from onset to admission to hospital was six months; and the average duration of illness from onset to death was seven and one-tenth months. The author appends a list of the principal symptoms and signs and laboratory findings of the disease, showing their relative frequency.

The Effect of Aluminium Hydroxide.

JOSEPH B. KIRSNER (*The American Journal of Digestive Diseases*, May, 1941) reports his observations on the effect of aluminium hydroxide on the acid-base balance and on renal function. His conclusions are based upon a study of 23 patients with peptic ulcer to whom aluminium hydroxide was administered. Three of these patients received more than 100 cubic centimetres daily for as long as 31 to 116 days; one patient received 393 cubic centimetres daily for 71 days. The period of observation in several cases exceeded seven to eight months, and the electrolyte balance was found normal in every case. The blood urea nitrogen and urea clearance tests for renal function were carried out in 14 cases of the series, and renal efficiency was not decreased in any instance in which it had been normal. The administration of aluminium hydroxide to three patients with a marked reduction in kidney function following alkalosis was followed by the maintenance of a normal acid-base equilibrium and by a gradual improvement in the urea clearance. The author concludes that aluminium hydroxide, even in massive doses, does not disturb the acid-base balance, that it does not impair renal function even when administered in large amounts over periods as long as seven to eight months, and that it may be given with safety to patients whose renal efficiency is considerably reduced.

Bronchiectasis.

ALAN JORDON OGILVIE (*Archives of Internal Medicine*, September, 1941) describes the natural history of bronchiectasis and reports the results of a study of this disease. The author presents an account of the anatomy and physiology of the normal bronchi, based on a study of the recent literature and on a dissection of a normal bronchial tree. The pathogenesis, the ætiology and the clinical and radiological features of the disease are described on the basis of an analysis of 68 cases, including a pathological examination of lobes excised at operation in 28 cases. A classification of the disease is suggested, which recognizes congenital cystic disease of the lung and atelectatic and bronchogenic types of acquired bronchiectasis. The author reaches the conclusion that bronchial dilatation may be temporary or permanent. Temporary bronchiectasis is always due to collapse of the lung tissue, whereas permanent bronchiectasis is the result of the interaction of many factors, of which the most important is infection of the bronchial wall. The numerous pathogenic factors in the evolution of bronchiectasis are grouped under two heads: (*a*) defects in the bronchial wall and (*b*) excessive strain and stress to which the bronchial wall may be subjected. The author states that no conclusive evidence is available that "congenital bronchiectasis" exists apart from congenital cystic disease of the lung. He emphasizes the value of an accurate history and careful physical examination in the diagnosis of bronchiectasis, and states that in but few cases are physical signs absent. The need for a detailed and extensive study of the prognosis in selected groups of cases is indicated. The causes of death in bronchiectasis are classified into three groups: (*a*) local exacerbation or spread of infection within the lungs or thoracic cavity; (*b*) direct complications of associated conditions and intercurrent disease; (*c*) dissemination of infection by the blood stream and the late results of chronic sepsis.

Pneumonia in Children.

CRAIG D. BUTLER *et alii* (*The Journal of the American Medical Association*, November 29, 1941) review 731 children admitted to Cook County Hospital from April, 1939, to May, 1940, in whom a diagnosis of pneumonia was confirmed. Of this total, 656 children (87·1%) received sulphapyridine, 19 (2·6%) received serum in addition to the drug, and 75 (10·2%) received no specific medication because they were moribund or convalescent on admission. The usual dose of sulphapyridine was one and a half grains per pound of body weight on the first day and three-quarters to one grain per pound on subsequent days. Children over six years of age received somewhat smaller doses, but at least one grain per pound the first day and half a grain per pound on subsequent days. The first two doses given were almost half of the calculated twenty-four hour requirement. Fluids given parenterally, inhalations of oxygen, and blood transfusions were administered freely when symptoms indicated their use. Specific antipneumococcus rabbit serum was given intravenously to patients not responding satisfactorily to chemotherapy after twenty-four to forty-eight hours of treatment. Of the 731 children studied, 54% were boys and 46% girls; 60% were white and 40% Negro; only 20% of the deaths occurred among Negro children. Forty-five *per centum* of the patients were classified as having bronchopneumonia and 55% as having lobar pneumonia. In the group aged one to two years, 68% had bronchopneumonia, whilst in the group over four years of age 72% had lobar pneumonia. The four types of pneumococci most frequently recovered were Types I, VI, XIV and XIX. Other organisms, notably streptococci and staphylococci, were identified in 17% of the cases. One noteworthy feature was individuality of Type I pneumococcus pneumonia. The patients with this type had more toxic symptoms; vomiting, abdominal pain, delirium and meningismus occurred more frequently. Empyema occurred eighteen times more often with this type than with all other forms of pneumonia. The mortality, however, was not greater than for the less severe forms. Suppurative *otitis media* occurred in 53 cases (7·4%). Empyema occurred in 29 cases or 4% of all that were studied, and in 18 cases (62%) of Type I pneumococcus pneumonia. In 18 cases the organism recovered from the empyema fluid corresponded to that previously recovered from the sputum. In the other 11 cases the organism differed. The authors consider that this indicates that the serum typing in this series was only 65% accurate. Among the 29 empyema patients, four (14%) died. There seemed to be no benefit from chemotherapy after the empyema was established. The average amount of sulphapyridine required to reduce the temperature to normal was 3·21 grammes and average total dose 6·17 grammes. Mild unfavourable reactions were encountered—vomiting in 17%, hæmaturia in 3·6%, and acute cerebral manifestations in 2%. No death could be attributed to the chemotherapy. Twenty of the 731 children died, with a resulting mortality of 2·73%; nine died within the first twenty-four hours of admission to hospital. All but one of the children who died were under four years of age. The mortality rate was 1·33% for the series after excluding the nine patients who were moribund on admission and two who were readmitted to hospital. The authors compare the figures collected by Abt in 1926 (15% mortality for all types of pneumonia) and those at the Cook County Hospital in 1938 (6·6% mortality for all types of pneumonia) prior to the specific therapy.

Vitamin Therapy.

N. JOLLIFFE (*The Journal of the American Medical Association*, November 1, 1941) discusses treatment of neuro-psychiatric disorders with vitamins. These disorders include polyneuritis in association with alcoholism, pellagra, beriberi or scurvy; Wernicke's syndrome, *delirium tremens*, Korsakoff's psychosis, some types of muscular dystrophy, of amyotrophic lateral sclerosis and of *paralysis agitans*. Improvement has been noted after vitamin treatment in these states. Sciatica, eighth nerve deafness, single nerve palsy, *herpes zoster*, hysteria, impotence and psychogenic pain are not included among the conditions in which vitamin therapy is indicated. Alpha-tocopherol and pyridoxin can be reasonably tried in muscular dystrophies, amyotrophic lateral sclerosis and *paralysis agitans*; there may be some transient objective benefit. In *paralysis agitans* the administration of 50 to 100 milligrammes of pyridoxin daily or every other day, by the intramuscular route, for three or four weeks, is associated with signs of some improvement. A full diet is given together with vitamin therapy.

The Asymptomatic Phase of Syphilitic Aortic Insufficiency.

WALSH MCDERMOTT, RALPH R. TOMPSETT AND BRUCE WEBSTER (*The American Journal of the Medical Sciences*, February, 1942) have studied the asymptomatic phase of syphilitic aortic insufficiency over the period from October, 1936, to April, 1941. They found that of 2,718 syphilitic patients examined at the syphilis clinic of the New York Hospital, 100 had syphilitic aortic insufficiency; nine had aneurysms as well. There were thus 91 patients who had the valvular defect alone. The patients were divided into three groups. Group I consisted of 45 patients who had no symptoms of cardiac insufficiency at the time of the original diagnosis. Group II consisted of 13 patients who admitted cardiac symptoms only after specific questioning, but had not sought medical advice because of these symptoms. Group III consisted of 33 patients who had symptoms related to their aortic disease. The authors emphasize that the classification of the patients into groups was made solely on the basis of the examiner's judgement at the time of the initial examination in the clinic; but few changes have occurred since their original classification. In order to avoid the placing of too much reliance on the patients'

statements alone, studies of the circulation were made. The vital capacity, arm-to-tongue circulation time and venous blood pressure were measured on the 47 patients belonging to the three groups who were available for clinical study; 23 were from Group I, seven from Group II, and 17 from Group III. The results showed a close correlation with the clinical impression of their cardiac status. The X-ray and fluoroscopic appearances of the aorta were consistent with normal width in 37 of 87 cases. In only three instances in a total of 135 did the Wassermann test when applied to the blood serum fail to produce a positive reaction in the absence of a history of previous antisyphilitic treatment. The authors conclude that aortic insufficiency due to syphilis is present in a clinically recognizable form for a period varying from two to ten years before the development of symptoms. This asymptomatic form of aortic valvular syphilis is encountered in approximately 50% of patients with valvular syphilis. Present-day prognostic data, since they are based on the course of the disease following the onset of symptoms of failure, are inapplicable to this large group of patients with cardiac vascular syphilis. There are no available data concerning the ultimate length of the asymptomatic phase; but from their study the authors think that it can be measured in months rather than years.

Acute and Chronic Nephritis.

F. D. Murphy and B. J. Peters (*The Journal of the American Medical Association*, January 17, 1942) have investigated the immediate results and the outcome after a period of ten years of the treatment of acute nephritis. They state that there is a great divergence of opinion regarding the ultimate prognosis of acute nephritis. Some authors hold that complete recovery usually occurs and that subsequent chronic glomerular nephritis is rare; others believe that chronic nephritis is a fairly common consequence. The authors deal with 205 patients suffering from acute nephritis who were studied in the initial stages of the disease and were kept under observation for periods varying from two to ten years afterwards. The series includes both adults and children. They are concerned particularly with eighty-nine patients who were specially studied. Of the 205 patients, 26 died in the acute stage, 35 did not return for reexamination (they were assumed to be cured) and the disease of 55 became chronic within a year. When these three groups are subtracted from the total of 205 a group of 89 remains. Of the 89 patients, 41 were considered cured, but six subsequently showed evidence of latent nephritis; 48 had some evidence of renal inflammation at the time of their discharge from hospital. The authors give some details of these 48 patients. They show that if the figures are corrected for those patients who did not return for reexamination, the percentage of the total of 205 who manifested latent or chronic nephritis was 43·5. They remark that there is no solution to the problem of how some patients who recover from acute nephritis are completely healed and others progress gradually to a state of chronic nephritis. The authors have been impressed by the fact that the lesion which failed to heal immediately tended to do so eventually if the patient rested and adhered to simple regulations of treatment. The failure to understand the significance of the latent phase is one of the main stumbling blocks in the recognition of the relationship between the acute and the chronic form. The intensity of the initial stage of acute nephritis has little or no bearing on the ultimate outcome, but the duration of the disease which precipitates the acute nephritis may often have an effect on the ultimate outcome of the renal lesion. The authors insist that the prognosis of acute nephritis is not so favourable as some authors have stated.

Paratyphoid Fever Treated with Sulphapyridine.

F. Muller (*The Lancet*, January 24, 1942) reports a case in which a man, aged twenty-seven years, suffering from paratyphoid fever, was treated with sulphapyridine. The clinical diagnosis was septicæmia caused either by *Bacterium paratyphosum B* or by the meningococcus. Sulphapyridine was given before a bacteriological diagnosis was made. The condition of the patient, who "looked toxic and gravely ill", was completely changed within thirty-six hours. The author points out that since paratyphoid fever occurs more often in a septicæmic form and is generally diagnosed earlier than typhoid fever, it is more suitable for chemotherapy. The leucopenia characteristic of the disease should be kept well in mind, in view of the tendency of the sulphonamide drugs to produce the same condition.

Thromboangiitis Obliterans and Dermatophytosis.

Mayer Naide (*The American Journal of the Medical Sciences*, December, 1941) puts forward evidence suggesting an ætiological relationship between dermatophytosis and *thromboangiitis obliterans*. He admits that the evidence is insufficient to enable a conclusion to be drawn. Certain clinical features of *thromboangiitis obliterans*, however, are more readily accounted for on the basis of fungous infection than by other ætiological agents so far proposed. The author thinks that in all cases of *thromboangiitis obliterans* a search should be made for dermatophytosis, and persistent treatment should be undertaken if it is found.

The Organic Phosphorus of Stored Blood.

Montague Maizels (*The Lancet*, June 7, 1941) has investigated the addition of glucose to stored blood with reference to organic phosphorus compounds. Guest has shown in 1932 that defibrinated blood stored at 37° C. shows a steady decrease in sugar content for some hours. When glycolysis ceases because sugar has all been used up, a rapid elimination of inorganic phosphates occurs from the organic phosphates of the erythrocytes. In the first investigation glucose was added to a sample of stored blood so that the final concentration was 1%. This was kept at 4° C. for ten to fourteen days and then compared with a control specimen which had had no sugar added. It was found that the addition of glucose caused a marked diminution in the breakdown of organic phosphates. The influence of glucose on phosphorolysis in cells separated from plasma was next investigated and at the same time the influence of hæmolysis on phosphorolysis was considered. The conclusion was that glucose had a marked inhibitory effect on phosphorolysis in the intact corpuscle, but none whatever on hæmolysed cells. It was also found that the addition of glucose did not alter appreciably the pH of the blood. It was thought that the inhibition of organic phosphorus breakdown is an aid in the survival of the erythrocyte.

Rheumatic Pneumonia.

Emanuel Z. Epstein and Edward B. Greenspan (*Archives of Internal Medicine*, December, 1941) present a study of the observations at autopsy in the lungs in 45 consecutive cases of acute rheumatic fever, selected regardless of the presence or absence of any clinical and gross pathological pulmonary changes. They were able to reconstruct the chronological sequence of events in the development of the pathological process in the lung from the purely microscopic stage, in which no clinical signs could have occurred, to the stage at which pulmonary damage was clinically manifest. They had the opportunity to study 14 cases of acute rheumatic fever, fatal in the initial attack, in which chronic cardio-valvular disease with chronic stasis in the lungs was absent. In some instances the entire course of illness lasted only a few days. In addition, 31 cases of acute rheumatic fever were studied in which one or more previous attacks had resulted in antecedent cardio-valvular disease with anatomical valvular changes. Here the combination of the effects of previous pulmonary stasis secondary to chronic valvular disease and the immediate effects of the acute and fatal attack were investigated. The first stage of the pulmonary changes consists of passage of considerable amounts of fluid into the alveoli, septa and interlobular spaces, with marked congestion of alveolar capillaries, engorgement and diapedesis of erythrocytes, together with desquamation of large mononuclear cells into the alveoli. This alveolitis is non-fibrinous and non-bacterial and strongly suggests alterations in the capillaries, with resultant increased permeability and diapedesis of erythrocytes and transudation of albuminous fluid into the alveoli. Hyaline membranes are formed in the respiratory bronchioles and the alveolar ducts in about 50% of the cases. As the disease is prolonged, this stage is followed by thickening and infiltration of the interlobular septa with mononuclear cells, with resultant fibrosis and organization. The smaller arteries show occasional thickening. Gross examination of the lungs at this stage reveals many patchy foci of dark red, fleshy, non-crepitant, non-aerated areas, which have a peculiar india-rubber consistency. This stage has been observed in 14 cases in which death was due to the initial attack of rheumatic fever and in which no previous valvular disease had existed. In all the remaining cases evidence of previous cardio-valvular disease with resultant pulmonary congestion was found, with the familiar effects of more or less long-standing stasis. But in many cases, in addition to the chronic stasis engendered by the previous attacks, the authors also observed the evolution of an acute stage similar to that described for the cases of fatal initial attack. And in nine cases typical hyaline membranes were seen. No Aschoff bodies were found in the lungs in any of the 45 cases. Twenty cases of chronic rheumatic cardio-valvular disease, mitral stenosis, in which the patient died of intercurrent disease or

of chronic heart failure without evidences of rheumatic fever, 23 cases of coronary arterial disease in which the patient died of heart failure, and 16 cases of hypertensive heart disease, with failure, were similarly studied to consider the possibility that the pulmonary lesions seen in cases of acute rheumatic fever were a result of the pulmonary stasis of congestive heart disease. In none of the control cases was a picture observed which was at all similar to that seen in cases of acute fatal rheumatic fever. The alveolitis and the formation of hyaline membranes are, in the authors' opinion, a result of an alteration in vascular permeability, with resultant diapedesis of erythrocytes and transudation of sero-albuminous fluid from the capillaries into the alveoli. This vascular damage has been found in cases of rheumatic fever in various other organs of the body. They have seen involvement of the main pulmonary artery and also, in several instances, severe inflammatory arteritis of the various branches of the pulmonary artery in the lungs in the cases presented. As further evidence of the tendency to vascular damage in rheumatic fever, resulting in increased permeability of the capillary wall, the authors have found intralobular hepatic œdema in 71% of the 45 cases of acute fatal rheumatic fever. In conclusion, they state that in their opinion a specific rheumatic lung or rheumatic pneumonia cannot be considered to exist. Aschoff bodies were not found in a single instance. A characteristic, though not specific, pulmonary picture is present, which consists of alveolitis, marked congestion, œdema, engorgement and the formation of hyaline membranes. These lesions are considered to be a result of damage to the capillaries, with resultant alterations in vascular permeability. Vascular damage was likewise present in the main pulmonary artery and its branches and in the hepatic capillaries. This vascular change corresponds to the involvement seen in the arterial tree in various organs of the body in acute rheumatic fever.

Prothrombinæmia and Analogues of Vitamin K.

ALBERT M. SNELL (*Journal of the Mount Sinai Hospital, New York*, July-August, 1941) discusses the analogues of vitamin *K* and their effect on prothrombinæmia. Vitamin *K* itself was found to be represented by the formula 2-methyl-3-phytyl-1,4-naphthoquinone. Substances with a quinoid nucleus were then studied with reference to their possible antihæmorrhagic properties. Phthiocol (2-methyl-3-hydroxy-1,4-naphthoquinone) was found to possess physical and biological properties similar to those of vitamin *K*. Even greater antihæmorrhagic properties were noted in 2-methyl-1,4-naphthoquinone, and in water-soluble 1,4-dihydroxy-2-methyl-3 naphthaldehyde and 4-amino-2-methyl-1-naphthol hydrochloride. As there are no important stores of vitamin *K* in the body, if the prothrombin level in the body is lowered suddenly for any reason, hæmorrhage will occur. If the compound 2-methyl-1,4-naphthoquinone cannot be given by mouth with bile salts, it should be given intravenously. In two hours after the injection any danger from prothrombin deficiency has been eliminated. The vitamin *K* analogues have been generally used by the staff of the Mayo Clinic. Walters and his associates reported that the mortality rate for all surgical procedures on jaundiced patients fell from 14·9% in 1937 and 1938 to 6·4% in 1939, when vitamin *K* analogues were used. In primary hepatic disease long-continued use of large doses of 2-methyl-1,4-naphthoquinone has been moderately successful and no toxic effects have been noted. Finally, the author advocates the routine use of naphthoquinone therapy to obviate unrecognized hypoprothrombinæmia of the newly born.

Experimental Sensory Neurone Degeneration.

W. M. WINTROBE *et alii* (*Journal of Clinical Investigation*, January, 1942) have shown that abnormal gait and degenerative changes in the peripheral nerves, spinal ganglia and posterior funiculi of the spinal cord develop in pigs reared on a diet deficient in factors other than thiamin, riboflavin, nicotinic acid and vitamins *A*, *D* and *C*. The authors found that abnormalities did not occur when the basal ration was supplemented with desiccated whole liver or brewer's yeast. The various fractions obtained during the manufacture of liver extract for the treatment of pernicious anæmia were assayed for their value in protecting the nervous system. The fraction used in the treatment of pernicious anæmia was the most effective of all those tested, but relatively large amounts were required. The value of the extract was not increased by parenteral administration. Attention is drawn to the possible relation of the degenerative changes in the nervous system of these pigs to those observed in some cases of pernicious anæmia. There was some evidence to suggest that the factors protecting the nervous system are distinct from the anti-anæmic substance.

Sodium Bicarbonate in the Treatment of Diabetic Ketosis.

LOUIS B. OWENS, JACKSON WRIGHT AND EDNA BROWN (*Archives of Internal Medicine*, December, 1941) have studied the value of the intravenous administration of sodium bicarbonate in diabetic ketosis. In 12 unselected cases of severe diabetic ketosis the usual treatment was begun. One thousand five hundred cubic centimetres of physiological sodium chloride solution were given on the patient's admission to hospital and repeated as necessary. Insulin was given hypodermically at the rate of 40 to 50 units an hour, until the level of sugar in the blood and urine approached normal limits. From then on the hourly dose of insulin was reduced according to the individual needs of the patient. Dextrose was administered when the level of sugar in the blood approached normal if acetone still persisted in the urine. Specimens of blood were taken and oxalated on the patient's admission to hospital and afterwards every three hours until the ninth hour; determinations of dextrose content, carbon dioxide combining power and ketone content were made on each specimen. In four cases the total output of urine was collected for periods of three hours and preserved for determination of ketone content. The dextrose in the blood was measured by the method of Somogyi, Shaffer and Hartman. The carbon dioxide combining power of the serum was estimated according to the method of Peters and Van Slyke, and the ketone content of the blood and urine was determined by the Van Slyke method with Denige's reagent. In nine of the 12 cases the intravenous administration of a 5% solution of sodium bicarbonate was started either immediately on the patient's admission to hospital or three hours afterwards; the remaining three cases were used as controls. The authors concluded that the intravenous injection of sodium bicarbonate raised the carbon dioxide combining power of the blood serum and relieved Kussmaul respirations, but was without effect on the level of sugar in the blood. No antiketogenic action was observed, but rather a slight tendency in some cases for the elimination of ketones to be delayed. The mortality rate was also studied in 154 cases of diabetic ketosis. The criteria used as a basis of selection of cases for this study were: evidence of uncontrolled diabetes severe enough to necessitate hospital treatment, the presence of sugar and acetone in the urine, an initial level of sugar in the blood over 200 milligrammes per 100 cubic centimetres and a carbon dioxide combining power of the blood serum under 30 volumes *per centum*. The 154 cases were divided into five groups according to the classification of I. M. Rabinowitch, A. F. Fowler and E. H. Bensley. In approximately one-third of the cases sodium bicarbonate was administered. The mortality rates of the treated cases and the controls were determined. The authors conclude that if the severity of diabetic ketosis is taken into proper consideration in each case, no difference is apparent between the mortality rates of those cases in which sodium bicarbonate was given and those in which no alkali was given. They consider that there is no practical basis for sodium bicarbonate therapy in the treatment of diabetic ketosis.

An Outbreak of Scurvy.

JULIA MORGAN AND A. S. GAULT (*Chinese Medical Journal*, August, 1941) discuss the condition of 132 flood refugees suffering from scurvy who were treated at Tsinan between December, 1935, and March, 1936. A definite picture of the disease was obtained, together with considerable information as to the efficacy of various forms of treatment. The disease was encountered in all grades of severity, from a slight pain and tenderness of one calf of a few days' duration with no objective signs to huge ecchymoses on both calves and thighs posteriorly, with brawny swelling, tenderness, limitation of movement on both flexion and extension, accompanied by extreme and disabling pain on any attempt to stand or walk. Painful swollen and bleeding gums were sometimes complained of, and were present in 86% of cases. The largest percentage of patients fell in the age groups 30 to 39, 40 to 49, 50 to 59, about 20% in each. The number of patients under twenty years of age was low, less than 10%, and there were no patients under twelve. Symptoms of scurvy appeared after three to eight months on a deficient camp diet, consisting of cereals and salt turnip; the addition of cabbage appeared to affect the incidence of scurvy, a lower incidence being present in the camps with the larger cabbage supply. Elevation of temperature was the rule in hospital patients with scurvy, and exaggeration of patella and biceps reflexes occurred in about half of the patients examined.

Special Abstract.

SOCIAL MEDICINE IN CHILE.

In the issue for January, 1942, of the *International Labour Review*, there appears an article on social medicine in Chile by Dr. Salvador Allende G., who is Minister of Health, Social Insurance and Assistance in that country. Readers may be reminded that the *International Labour Review* was originated by the League of Nations and is published every month by the International Labour Office, which now has its headquarters at McGill University, Montreal, Canada. Some of the facts set out and the views stated in this review are of such interest that a short abstract has been prepared for the information of Australian practitioners.

The author opens with the remark that he has set out the policy followed in Chile in regard to social medicine not merely as a statement of facts and of plans, but also in the belief that what he calls the biosocial protection of the working masses of a country constitutes the surest foundation for its defence and, he adds, for the defence of the American continent. In Latin-American countries human capital has been rather neglected, and the author holds that public opinion should be stimulated so that all the reserves of the Latin-American countries can be used for the protection and needs of that human capital. A low standard of life means a decline in working capacity, and though it is not realized this is in effect equivalent to a strike among workers. If conditions of life are to be improved, and if there is to be more scope for the creative capacity and the spiritual sources of mankind, two methods seem to the author to be indispensable. The first of these is international in character and the second has to do with the organization of social security.

In regard to the international method, the author points out that smaller countries are in the habit of obtaining credits from more highly developed States for the purposes of national defence or for the development of particular industries. He thinks that credit should be granted in order to raise the standard of living of the masses in the Latin-American countries, in order to promote public health, to improve the sanitation of the towns and to provide for the workers, mothers and children. This would establish a new sense of interrelationship and economic solidarity, and would not only strengthen the links between poorly equipped and well-equipped countries, but would hold the promise of a new structure in the international field. This structure would give evidence that totalitarian propaganda was wrong and that the three Americas were being brought more closely together to achieve a new end; this end is the protection of the human capital which is a basis of the greatness of the American nations. In regard to organized social security, social legislation must be so planned that it will cover the worker and his family as a unit—the worker from the medical and economic standpoints, and the family from the medical standpoint.

The Economic and Social Background.

Chile forms a long and narrow stretch of land extending north and south between the Cordillera of the Andes and the Pacific Ocean. Its greatest width is 280 miles and its narrowest 94. The total area is 290,000 square miles. The climate is maritime in character and there are no great differences between the summer and winter temperatures. In the northern zone rain is rare and in the south frequent and abundant. The country can be divided into five zones, as follows: (*a*) a mining zone in the north; (*b*) a mixed mining and agricultural zone; (*c*) a mixed agricultural and industrial zone, composed of the provinces of Valparaiso and Santiago; (*d*) an agricultural zone, which is the largest of the five; (*e*) a forest and stock-raising zone stretching down to Cape Horn.

The Population.

The population of the country, according to the 1940 census, is 5,100,000, giving a population density of 17 per square mile. It may be noted here that the population density of Australia is 2·37 persons to the square mile; it is the most sparsely populated of the civilized countries of the world. For the other continents the number of persons to the square mile is as follows: Europe, 120; Asia, 73; Africa, 13; North and Central America, 21; South America, 13. About half the population of Chile is rural and is engaged chiefly in agricultural pursuits, and one-quarter lives in the industrial zone. The population is white and homogeneous, and its growth is due entirely to natural increase, without any contribution from immigration. Only 2·5% of the total number of inhabitants are foreigners, the majority in declining order of importance being Spaniards, Italians, Englishmen, Germans, North Americans and Latin-Americans. Of the total number of persons in the country, 3,100,000 fall within the working age of fifteen to sixty years.

Vital Statistics.

The general mortality rate, including infant mortality, for the last ten years is 25 per thousand inhabitants. Infantile mortality in recent years has been 240 (deaths under one year) per thousand live births, a rate naturally connected with the high birth rate, which is 34 per thousand inhabitants. At this point it may be indicated that according to the "Official Year Book of the Commonwealth of Australia" for 1940 the rate of infantile mortality for children in Australia below one year of age in the year 1939 was 38·23 per thousand live births. On the other hand, the birth rate in Australia was only 17·65 per thousand of the mean population for 1939. The author points out that since 1937-1938 (this apparently is the year providing these figures) the infantile mortality in Chile has declined in consequence of measures taken by the Government. These measures include the infant welfare services of the Workers' Compulsory Insurance Fund and the comprehensive medical assistance provided by the unified services in the south of the country. Infantile mortality is largely due to gastro-intestinal disorders and respiratory infections. Tuberculosis, with a death rate of 245 per 100,000, accounts for 17% of the general mortality. This is followed by infectious and contagious diseases with 60 per 100,000 (typhoid fever 13) and cardio-renal affections due mainly to syphilis, which are responsible for 11% of the general mortality.

Wages.

Wages vary substantially from one area to another, being higher, for example, in the mining zone than in the agricultural zone. Generally speaking, however, the greater part of wages is devoted to the provision of food and housing, only a small margin being left for clothing and other necessities.

Departments of the Ministry of Health.

The Ministry of Health, Social Insurance and Assistance has three departments. The first is the General Social Assistance Department. It controls and supervises 106 hospitals and sanatoria with 21,000 beds and 47 health centres in rural areas. There are 1,000 medical practitioners on its staff. The second department is the Public Health Department, which is under a general director and is responsible for the supervision of infectious and contagious diseases, industrial hygiene, international hygiene, inoculation and sanitary conditions in general. The third department, the Social Insurance Department, is responsible for the control of the insurance and provident funds.

Under the Social Insurance Department there are 30 insurance and provident funds. The author has some comments to make on the four most important. The Workers' Compulsory Sickness, Invalidity and Old Age Insurance Fund has a membership of 1,200,000. This fund covers the economic risks of sickness and the cost of preventive medicine (including periodical medical examination) and curative action. It also covers the medical and economic risks of total invalidity, the risk of old age and the cost of maternity and infant welfare until the child reaches the age of two years. The fund is financed by contributions amounting to 2% of their wages paid by the workers, 5% paid by the employers and 1·5% paid by the State.

There are private employees' funds which have 220,000 members. Each employee has to accumulate savings at the rate of 18·13% of his salary, 5% being paid by the employee and 13·33% by the employer. These funds provide for periodical medical examination for the diagnosis of tuberculosis, cardio-vascular disease and syphilis in particular, and they also assist members to acquire a home, the payment for which is spread over a considerable period.

The Public Employees' and Journalists' Fund has 60,000 members. It is a fund similar to those of the private employees. In addition, it provides pensions after thirty years of service and pensions for widows and orphans, provided that contributions have been paid for a period of ten years.

There are funds for the armed forces. They have 30,000 members and the benefits are similar to those provided by the Public Employees' Fund.

The risks of industrial accidents and occupational diseases are met by the payment of premiums by the employers at the rate of two to six per thousand on their wage bills.

The Ministry of Education.

There are three schools of medicine. Approximately 100 students qualify every year and there are 2,700 doctors in the country. There are two schools of dentistry, two schools of chemistry and pharmacy, five schools for hospital nurses, one school for district nurses and three for women district visitors. Finally, the State University of Chile organizes each year two or three post-graduate courses, and the national institutions responsible for public health meet the cost of travelling and residence in Santiago involved.

Government Policy.

The progress that has been made in recent years in the field of medicine and health organization has not caused any great improvement in the general health conditions during the last thirty years. The protection and improvement of public health are therefore considered by the Government to be of fundamental importance. In these circumstances it has made efforts for the protection of labour and wages and to provide housing. The housing shortage is estimated at about 150,000 dwellings. To cope with this deficiency a special building scheme has been brought into operation, and it is hoped that this will be completed in ten to fifteen years. Particular attention has been paid to the production of milk, and a large number of pasteurizing plants have been erected.

The Ministry of Health Activities.

The activities of the Ministry of Health, Social Insurance and Assistance are described as having four chief aspects: chemical and pharmaceutical policy, the standardization and improvement of health services and social assistance, the extension of social insurance and legislative action in general. In regard to the first of these, it is pointed out that the outbreak of war raised a serious problem, in that the prices of medicaments rose and their importation became difficult. A Prices and Drugs Committee was appointed to control prices and to enable laboratories to obtain a supply of raw materials. A new plant has been purchased for a bacteriological institute, which will increase the production of vaccines, different forms of serum and other products. The Chile Laboratory, which belongs to the Workers' Compulsory Insurance Fund, has also been extended.

In regard to social assistance (this includes the management of 106 hospitals), the economic situation of the staff has been improved; the sum allocated to the feeding of patients has been substantially increased; more X-ray apparatus has been purchased, and new clinical laboratories have been set up; and a policy is being pursued by which full-time service from medical practitioners is being secured. On the public health side a system for the training of specialized technicians has been instituted. A bill is under consideration by the Chamber of Deputies to bring about certain changes in the Workers' Compulsory Insurance Fund. The new scheme will be financed by a contribution from workers, employers and the State, and will provide curative and preventive medical protection for 1,200,000 workers and for the 2,800,000 members of their families. This protection will extend from a system of periodical medical examination to the special care of mother and child in confinements and the medical care of pensioners; there will also be a payment of cash benefit for temporary incapacity due to sickness, industrial accident or maternity. On the economic side the new measure will provide for the payment of pensions in case of partial or total invalidity, old age and death. The number of persons who will be entitled to such pensions is estimated at 320,000.

The *Preventive Medicine Act* provides for the diagnosis at an early stage of the three main factors in Chilean morbidity—syphilis, tuberculosis and cardio-vascular disease. Of 223,000 persons examined in three years, 78·5% were free of the diseases, 9% suffered from syphilis, 7·1% from tuberculosis and 5·4% from cardio-vascular disease. Bills have been introduced for the establishment of a school of hygiene and a nutrition institute, for the making of venereal contagion a criminal offence, for the control of the importation of narcotics and for the unification of the medical services of the Workers' Compulsory Insurance Fund and of the social assistance authorities as a logical extension of the granting of medical benefit to the workers' families.

A Health Protection Loan.

The author of this report points out that the policy so far described is in complete agreement with the most up-to-date and practical principles of health organization; but he adds that even this is not considered sufficient by the Government. It is proposed in addition to secure a loan of 20,000,000 United States dollars "for the execution of a health protection programme". The author sums up the views of the Chilean Government:

The Chilean Government believes that any possibility of greatness for the nation must be based on a strong, enlightened and healthy people. It also believes that national defence depends primarily on good biological conditions for the inhabitants of the country. Lastly, it believes that an effective contribution will be made to continental defence if cities are made more healthy, chemical and pharmaceutical laboratories are developed, and the country is given the sanatoria, hospitals, rest homes and other equipment for making biological protection effective.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 94, of March 26, 1942.

NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

His Excellency the Governor-General in Council has approved of George Bell, O.B.E., M.B., Ch.M., F.R.A.C.S., and Archibald James Collins, D.S.O., M.C., M.B., Ch.M., being granted the honorary rank of Surgeon Commander, dated 1st December, 1941.—(Ex. Min. No. 24.)

AUSTRALIAN IMPERIAL FORCE.

Australian Army Medical Corps.

The undermentioned officers relinquish temporary ranks where held, are seconded in their units of the Citizen Military Forces, and are appointed to units of the Australian Imperial Force as from the dates and in the ranks indicated, with regimental seniority in accordance with their Army seniority:

To be Major.—Captain (Temporary Major) R. Mackey, 15th January, 1942.

To be Captains.—Captain (Temporary Major) C. R. Morton, 16th January, 1942; Captains I. W. McNaught, 8th December, 1941; L. W. Martin, 22nd December, 1941; K. A. Barr, 8th December, 1941; and H. I. McKenzie, 15th January, 1942.

To be Captains.—Captains G. M. Brodie, 8th December, 1941; and S. D. Foley, 12th December, 1941.

The undermentioned officer is appointed from the Reserve of Officers to a unit of the Australian Imperial Force as from the date and in the rank indicated:

To be Captain.—Honorary Captain A. S. Jackson, 15th January, 1942.

The undermentioned are appointed to units of the Australian Imperial Force as from the dates and in the ranks indicated:

To be Captains and are seconded.—James Murray McCracken, Joseph Paxton Robinson, David Philip Churton, Beric Tristram Glanville-Hicks, David Gordon Phillips and Ronald William Edward Hoyling, 2nd December, 1941; Nairne Guthrie Elder, Geoffrey Gossip Harkness, John Forrest Davies and Alan Ross Wakefield, 18th December, 1941; Keith John McInnis King, Kenneth John Shellshear, Douglas Seavington Stuckey, Alun Iorwerth Rhydderch and Lynn Harvey Joseph, 20th December, 1941; Bruce Henry Peterson, 3rd January, 1942; Norman Reginald Scott-Young, Arthur Lavender Stephenson, Roland Adrian Glennie Holmes, Peter Howard Greenwell and Ralph Beattie Blacket, 6th January, 1942; Ronald Victor Pratt and Edward Joseph McDonald, 7th January, 1942.

The following changes *et cetera* are made:

To be Lieutenant-Colonel.—Major R. J. A. Henderson and to command a Light Field Ambulance, 18th December, 1941.

Major (Temporary Lieutenant-Colonel) J. B. D. Galbraith to command (temporarily) a Light Field Ambulance, and retains the temporary rank of Lieutenant-Colonel, 22nd December, 1941.

To be Majors (Temporarily).—Captains J. C. Stewart, 1st January, 1942, and C. W. Furner, 5th January, 1942.

The undermentioned officers are transferred from Reinforcements: Captains M. C. Newland, 17th December, 1941; A. M. Barron, 24th December, 1941; D. B. Sillar, 17th December, 1941; J. Gribben and E. J. T. Giblin, 17th December, 1941; M. J. M. Lapin, 24th December, 1941;

I. A. N. McCallum, 17th December, 1941; S. D. Foley, 5th January, 1942; M. G. Gratton and J. G. McMahon, 17th December, 1941.

To be Brigadier (Temporarily).—Colonel W. W. S. Johnston, D.S.O., M.C., E.D., 10th August, 1941.

To be Lieutenant-Colonels (Temporarily).—Majors M. A. Rees, 28th October, 1941; J. M. Dwyer, H. M. Fisher, F. R. Hone, I. J. Wood and J. O. Smith, 10th November, 1941; and E. S. J. King, 25th November, 1941.

To be Majors (Temporarily).—Captains A. Fryberg, 28th September, 1941 (in lieu of notification respecting this officer which appeared in Executive Minute No. 134/1941, promulgated in *Commonwealth Gazette* No. 266 of 18th December, 1941); A. K. Green, 28th October, 1941; R. Warden, W. D. Refshauge, A. C. Mendlesohn, E. P. Cherry and W. M. Irwin, 22nd November, 1941; N. J. Bonnin, T. H. Ackland, R. W. Johnson, N. H. Robinson and R. Officer, 25th November, 1941.

Australian Military Forces.

Australian Army Medical Corps.

Northern Command.

First Military District.

Honorary Captain S. J. Rumbold is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 12th February, 1942.

To be Honorary Captains.—John Joseph Ward and Lynn David Walters, 23rd February, 1942.

The date of appointment from the Reserve of Officers of Honorary Captain (now Captain (provisionally)) K. B. Shallard, notification of which appeared in Executive Minute No. 20/1942, promulgated in *Commonwealth Gazette* No. 28 of 1942, is amended to read "5th January, 1942".

To be Majors (temporarily).—Captains (provisionally) W. J. Arnold, J. R. Adam, R. S. Lahz and J. A. Lynch, 24th February, 1942.

The notification respecting the date of appointment of Captain (provisionally) K. B. Shallard, which appeared in Executive Minute No. 44/1942, promulgated in *Commonwealth Gazette* No. 70 of 1942, is withdrawn.

Eastern Command.

Second Military District.

To be Majors (temporarily).—Captains N83382 F. H. Callow, N57472 T. E. Y. Holcombe, N83372 E. H. Hipsley and H. McL. Hollingworth, 16th February, 1942.

To be Captain (provisionally).—Ross Macpherson Dunn, 27th February, 1942.

To be Honorary Captains.—Max Robert Morey, Mervyn William Matheson, 16th February, 1942, Horace Joseph Eizenberg and Albert Stuart Bradbridge Studdy, 24th February, 1942.

Major (Temporary Lieutenant-Colonel) N. H. Meacle is appointed to command a Casualty Clearing Station, 14th January, 1942.

Honorary Captain H. E. Marsden is appointed from the Reserve of Officers and to be Captain (provisionally), 7th February, 1942.

To be Major (temporarily).—Captain N105513, N. Rau, 25th February, 1942.

To be Captain (provisionally) and Major (temporarily).—Harry John Daly, 1st March, 1942.

To be Captains (provisionally).—Edward Mansfield, Francis Henry Hales Wilson, John Richard Kay-Mouat, George Arthur Williams Johnston, 3rd March, 1942, Stanley Clive Maynard, 4th March, 1942, William Morton Craig MacDonald, 6th March, 1942, Hilary James Kenny and Leslie Ewart Senis Larbalestier, 7th March, 1942.

To be Honorary Captain.—William Frederick James Cammack, 24th February, 1942.

Southern Command.

Third Military District.

Honorary Captain F. G. Middleton is retired, 15th December, 1941.

To be Honorary Lieutenant-Colonel.—Sir Hugh Berchmans Devine, 6th March, 1942.

The provisional appointments of Captain (Temporary Lieutenant-Colonel) C. W. Ross, Captains (Temporary Majors) W. I. E. Mackenzie and M. O. K. Hughes and Captains K. H. Macfarlane, C. M. Greer, G. Forsyth, W. J. Long and N. A. Longden are confirmed.

To be Major (temporarily).—Captain (provisionally) R. J. Farnbach, 6th February, 1942.

Lieutenant-Colonel H. McLorinan is transferred from Australian Army Medical Corps, Australian Imperial Force, and to be Major and Honorary Lieutenant-Colonel, 8th November, 1941.

The notification respecting the appointment of Honorary Captain S. G. Lauricella, which appeared in Executive Minute No. 134/1941, promulgated in *Commonwealth Gazette* No. 149 of 1941, is withdrawn.

Fourth Military District.

Honorary Captain S36298 S. E. Holder is appointed from the Reserve of Officers and to be Captain (provisionally), 2nd January, 1942.

Sixth Military District.

Major C. G. Thompson, V.D., is appointed from the Reserve of Officers, 16th January, 1942.

Western Command.

Fifth Military District.

The notification respecting the appointment of Captain (provisionally) W16416 G. L. Myles which appeared in Executive Minute No. 212/1941, promulgated in *Commonwealth Gazette* No. 247 of 1941, is withdrawn.

To be Honorary Captain.—Jean Wilmore Ainslie, 26th January, 1942 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 40/1942, promulgated in *Commonwealth Gazette* No. 63 of 1942).

Captain (provisionally) W. A. Hillman is retired, 16th February, 1942.

Correspondence.

CONTROL OF MEDICAL EQUIPMENT.

Sir: The Medical Equipment Control Committee has placed Cyclopropanum in class C, that is, "Drugs not considered essential and which do not justify importation or manufacture".

I wish to protest against this classification and support the letter of Dr. Renton which appeared in The Medical Journal of Australia, February 28.

Cyclopropane is the biggest advance in anæsthesia that has been introduced in the last ten years. It supplies that long-felt want: a gas anæsthetic which will give relaxation without heavy premedication and at the same time allow oxygen in a proportion up to 85%. With this anæsthetic there is no fear of cyanosis nor anoxæmia and it is the safest anæsthetic for most of the very seriously handicapped patients.

It has the further advantage that it is supplied in very small cylinders and not in the size that is so urgently required by the army.

Yours, etc.,

Gilbert Brown.

Adelaide,
March 18, 1942.

INDICATIONS FOR THE INTRAVENOUS ADMINISTRATION OF FLUID.

Sir: In your issue of March 21, 1942, page 357, under the heading of "Indications for the Intravenous Administration of Fluid", the Blood Transfusion Committee of the New South Wales Commission of the Australian Red Cross Society discusses the phenomena of shock.

Reference is made to various symptoms and signs, and much stress is laid on low blood pressure, circulatory failure, and fall in blood volume. No direct reference, however, is made to the fundamental phenomena of secondary shock, namely, loss of plasma colloids without loss of formed elements, and consequent increase in blood viscosity and hæmo-concentration. Hæmo-concentration occurs long in advance of any fall in blood pressure, and the degree of shock should be estimated on this basis and the blood pressure relegated to its position of very minor importance.

Moon (*Annals of Surgery*, Volume CX, pages 260-272) demonstrates very clearly the necessity for plasma or serum transfusion for shock and the embarrassment likely to result from whole blood transfusion in this condition. In one of his graphs, showing the curves of hæmo-concentration and blood pressure in experimental shock, he demonstrates that hæmo-concentration develops immediately, reaching 15% four hours later, in which time the blood pressure was above 140. Blood reaches its maximum concentration four hours before the blood pressure falls

to a critical level. Estimations of blood pressure, therefore, seem to be a fallacious guide to the degree of shock, which should be estimated by either a blood viscosity estimation or a simple and reasonably reliable basis, the hæmoglobin concentration, the alterations in which run very closely parallel with the degree of shock.

In two recent cases of very severe burns, treated by serum transfusion in the Brisbane Hospital, one being saved who would otherwise have most certainly died, the hæmo-concentration developed very rapidly even while the patient's general condition, if estimated by the blood pressure, was fairly good. Quarter-hourly checks of hæmoglobin showed a rise on two occasions, from 110% to 120% in about twenty minutes (100% = 14·5 grammes *per centum*). The hæmoglobin estimation can also be used as a rough guide to the amount of serum to be transfused immediately. The middle digit of the hæmoglobin percentage, divided by 2, gives the amount in litres needed by the patient.

In conclusion I should like to point out that a serum transfusion by gravity alone is very difficult owing to the high viscosity of the blood in secondary shock, so that considerable time may be wasted by attempting to get the serum from a simple irrigator. Some kind of pressure system is absolutely necessary, as has been found by experience in the Brisbane Hospital.

By the plasma and serum transfusions we have at our command, for the first time in the history of this country, the only rational means of treating secondary shock.

Yours, etc.,

J. V. DUHIG,

Professor of Pathology.

The University of Queensland,
Brisbane,
March 27, 1942.

INTRADERMAL INOCULATION.

SIR: It is well recognized that the usual procedure for inoculation against typhoid and paratyphoid results in considerable disability among service personnel.

Following upon a discussion of this subject with Professor Platt, of Adelaide University, an attempt was made to improve results by securing a slower absorption of the vaccine than is attained by the usual method.

The hypodermic needle is inserted tangentially into the dermis, and owing to the greater tissue tension, the forefinger and thumb of the left hand are used to prevent the detachment of the needle by the higher pressure required for the injection. As the white wheal develops on injection the needle is pressed further into the separating layers of the dermis. The skin above becomes white with "orange skin" depressions. Using this method, less than 3% of those injected suffered any considerable inconvenience.

It is hoped to indicate at a later date the degree of immunity resulting.

Yours, etc.,

R. FRANCIS MATTERS,

District Naval Medical Officer, Adelaide.

Adelaide,
March 23, 1942.

National Emergency Measures.

MEDICAL EQUIPMENT.

THE Medical Equipment Control Committee desires to direct the attention of all members of the medical profession to the following information.

Formalin and Permanganates.

Available supplies of formalin are very urgently required for defence purposes and must be conserved. It is necessary also to limit the use of permanganates to essential purposes. Both these materials are used in appreciable quantities for disinfection which in many instances could be carried out by other means. It will not be practicable to make supplies of formalin and permanganates available for disinfection purposes except where it can be certified that other methods of disinfection cannot be employed.

It is asked that careful supervision of disinfection measures be exercised and that formalin and permanganates be not used in any circumstances where some alternative method of disinfection can be employed.

Medical Prizes.

THE STAWELL PRIZE.

THE Medical Secretary of the Victorian Branch of the British Medical Association announces that the Stawell Prize, a memorial to the late Sir Richard Stawell, is open for competition. The amount of the prize is £30. The conditions are as follows:

1. The prize shall be awarded to the writer of the essay adjudged to be the best on a subject selected annually.
2. The subject for 1942 is "The Treatment of Peptic Ulcer".
3. The dissertation should be based on personal observation and experience of the writer.
4. The competition is open to graduates of any Australian university.
5. The trustees reserve the right to withhold the award.
6. Essays must be delivered to the Medical Secretary, British Medical Association (Victorian Branch), by 4 p.m. on July 31, 1942.
7. Each essay must be typewritten or printed, and must not exceed 75,000 words in length.
8. Each essay must be distinguished by a motto and must be accompanied by a sealed envelope marked by the same motto, containing the name and address of the author.
9. The trustees reserve the right to publish the prize essay.

Obituary.

GEORGE ERNEST CLEMONS.

DR. W. KEVERALL MCINTYRE pays the following tribute to the memory of the late Dr. George Ernest Clemons.

It is a great privilege to be able to pay a personal tribute to the memory of Dr. G. E. Clemons, who died in Launceston on February 5, 1942.

We had been close friends for the last twenty years. He was a very keen sportsman with a great love of the country, and we spent many happy days camping and shooting together.

When I began to practise in Launceston soon after the last war he showed me many great kindnesses, offering to assist me in practice and to help at operations. It can be readily understood what this meant to a younger man beginning practice, who had only had war experience since graduation, and full advantage was taken of his offer over many years.

There is no need for me to dwell on his professional ability as a surgeon and physician. He belonged to the older school, before specialization became so general. This gave him a very wide experience, and as he was an omnivorous reader of all current journals and had an exceptionally retentive memory, he was invaluable as a consultant. When camping he always took an anatomy and pathology book with him, and would revise a chapter or two before turning in, and this lifelong habit of revision and keeping abreast of modern teaching made it difficult to trip him up or beat him in an argument on a professional subject. His opinion was thus always well balanced and extremely helpful, and it has been a great relief on many occasions to share responsibility with him in this way.

He was seventy-six when he died, and within a few days of his death he asked me to get him a journal from the library, as he wished to read an article to which he had seen a reference. The next day, although he found it difficult to speak, he gave me a very trenchant criticism of the opinions expressed in the paper.

One of his most attractive qualities was his quiet, dry sense of humour.

Besides his professional comrades, many thousands of people to whom he has been counsellor and friend as well as doctor, will feel his loss very deeply.

DONALD JAMES SHALE.

WE are indebted to Dr. Howard Williams for the following appreciation of the late Captain Donald James Shale.

My first acquaintance with Donald Shale was during his term of residency as a medical officer at the Children's

Hospital, Melbourne, during the year 1941. He finished his appointment and joined the Australian Imperial Force in October, 1941. The same keenness, enthusiasm and thoroughness which had enabled him to complete a very brilliant medical course at the Melbourne University characterized his clinical work at the Children's Hospital. In addition to his medical ability, Don had a very lovable and upright character. Sir William Osler in one of his lectures stated that the best test of a man's character was the respect in which he was held by his professional brethren; Donald Shale stood high in the respect of his medical colleagues. There is no doubt that with his medical ability and his character Donald Shale would have been a leader in his profession. To his wife and the members of his family circle we offer our deepest sympathy.

RICHARD SANDERS ROGERS.

WE regret to announce the death of Dr. Richard Sanders Rogers, which occurred on March 31, 1942, at Adelaide, South Australia.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Sands, John Robert, M.B., B.S., 1941 (Univ. Sydney), Royal Prince Alfred Hospital, Missenden Road, Camperdown.

Marsh, Stafford Howie, M.B., B.S., 1939 (Univ. Sydney), 65, Belgrave Street, Kempsey.

Kidd, Donald Stuart, M.B., B.S., 1941 (Univ. Sydney), Sydney Hospital, Macquarie Street, Sydney.

Notice.

A MEETING of the clinical section of the Medical Board of the Royal Prince Alfred Hospital will be held on Monday, April 13, 1942, in the A2 lecture theatre of the hospital at 4.30 o'clock p.m. The meeting will take the form of clinical demonstrations. Dr. E. M. Fisher will give a demonstration of cases of carcinoma of the rectum, and members of the ear, nose and throat department will also give a demonstration. Medical practitioners are invited to be present.

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned has been registered, pursuant to the provisions of *The Medical Act*, 1939, of Queensland, as a duly qualified medical practitioner:

Williams, Frederick James, M.B., B.S., 1921 (Univ. Melbourne), District Hospital, Bowen.

Medical Appointments.

Dr. Lindon Archdall Langley has been appointed, on probation for a period of twelve months, a Medical Officer of the Department of Mental Hygiene, Victoria, under the provisions of the *Public Service Act*, 1928.

Dr. Robert Harvey Leeds has been appointed Government Medical Officer at Biloela, Queensland.

Dr. Frederick James Williams has been appointed coordinating officer for the hospital, ambulance and first aid services in the civil defence organization in the town of Bowen, Queensland.

Dr. Basil Lloyd Hart has been appointed a trustee of the Boys' Grammar School, Ipswich, Queensland, in pursuance of the *Grammar Schools Act*, 1860.

Dr. Benjamin Gilmore Wilson has been appointed a trustee of the Boys' Grammar School, Ipswich, Queensland, upon nomination of the subscribers, in pursuance of the *Grammar Schools Act*, 1860.

Books Received.

"Aids to Obstetrics", by L. Williams, M.D., M.S. (London), F.R.C.S. (England), F.R.C.O.G.; Eleventh Edition; 1942. London: Baillière, Tindall and Cox. Foolscap 8vo, pp. 246, with 10 illustrations. Price: 4s. net.

"Disease and the Social System", by A. Guirdham, M.A., D.M., B.Sc. (Oxon), D.P.M. (London); 1942. London: George Allen and Unwin Limited. Medium 8vo, pp. 256. Price: 10s. 6d. net.

"Disorders of Blood Pressure", by various authors, edited by Sir H. Rolleston, Bt., G.C.V.O., K.C.B., M.C., F.R.C.P., and A. Moncrieff, M.C., F.R.C.P.; 1942. London: Eyre and Spottiswoode Limited. Medium 8vo, pp. 93, with 9 illustrations. Price: 6s. net.

Diary for the Month.

APR. 14.—Tasmanian Branch, B.M.A.: Branch.
APR. 15.—Western Australian Branch, B.M.A.: Branch.
APR. 17.—Queensland Branch, B.M.A.: Branch.
APR. 24.—Queensland Branch, B.M.A.: Council.
APR. 24.—Tasmanian Branch, B.M.A.: Council.
APR. 30.—New South Wales Branch, B.M.A.: Branch.
APR. 30.—South Australian Branch, B.M.A.: Branch.
MAY 1.—Queensland Branch, B.M.A.: Branch.
MAY 6.—Western Australian Branch, B.M.A.: Council.
MAY 7.—South Australian Branch, B.M.A.: Council.
MAY 8.—Queensland Branch, B.M.A.: Council.
MAY 12.—Tasmanian Branch, B.M.A.: Branch.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

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Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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